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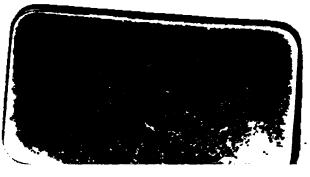

MEDICO-CHIRURGICAL TRANSACTIONS.

PUBLISHED BY

THE ROYAL

MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON.

VOLUME THE THIRTY-SEVENTH.

LONDON:
LONGMAN, BROWN, GREEN, AND LONGMANS,
PATERNOSTER-ROW.

1854.

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AUGUST 1854.

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1818 Thomas Addison, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; New-street, Spring-gardens. C. 1826. V.P. 1837. P. 1849. C. 1853.

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1828 WILLIAM COULSON, Surgeon to the Magdalen Hospital, Consulting Surgeon to the City Lying-in Hospital, and Senior Surgeon to St. Mary's Hospital; Frederick's-place, Old Jewry. C. 1831. L. 1832. V.P. 1851.

1817 *SIR PHILIP CRAMPTON, Bart., F.R.S., Surgeon-General to the Forces in Ireland; Dublin.

1841 MERVYN ARCHDALL NOTT CRAWFORD, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; Upper Berkeley-street, Portman-square. C. 1853.

1822 SIR ALEXANDER CRICHTON, M.D. F.R.S. and F.L.S., Physician in Ordinary to their Imperial Majesties the Emperor and Dowager Empress of all the Russias; the Grove, Sevenoaks, Kent. C. 1823.

1847 GEORGE CRITCHETT, Assistant-Surgeon to the London Hospital, and the Royal London Ophthalmic Hospital; Finsbury-square.

1837 JOHN FARRAR CROOKES, Russell-square.

1849 *WILLIAM EDWARD CROWFOOT, Beccles, Suffolk.

1851 JAMES CAMERON CUMMING, M.D., 1, Cadogan-place, Sloane-street.

1818 WILLIAM CUMING, M.D., Professor of Botany at the Glasgow Institution, and Surgeon to the Royal Infirmary at Glasgow.

1846 HENRY CURLING, Surgeon to the Royal Sea Bathing Infirmary; Ramsgate, Kent.

Elected

1837 THOMAS BLIZARD CURLING, F.R.S., *Treasurer;* Surgeon to, and Lecturer on Surgery at, the London Hospital; New Broad-street, City. S. 1845. C. 1850.

1847 JOHN EDMUND CURREY, M.D., Lismore, Ireland.

1836 GEORGE CURSHAM, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, and to the Female Orphan Asylum; Savile-row, Regent-street. S. 1842. C. 1850.

1822 CHRISTOPHER JOHN CUSACK, Chateau d'Eu, France.

1852 THOMAS CUTLER, M.D., Physician to the Spa General Dispensary; Spa, Belgium.

1828 ADOLPHE DALMAS, M.D., Paris.

1851 NATHANIEL JOHN DAMPIER, Surgeon to the Farringdon General Dispensary and Lying-in Charity; Woburn-place, Russell-square.

1836 *JAMES STOCK DANIEL, Ramsgate.

1850 JOHN BAMPFYLDE DANIELL, M.D., Physician to the Royal Pimlico Dispensary; Grosvenor-street, Grosvenor-square.

1820 GEORGE DARLING, M.D., Russell-square. C. 1841.

1818 *SIR FRANCIS SACHEVEREL DARWIN, Knt., M.D., Breadsall Priory, near Derby.

1848 HENRY DAUBENY, 34A, York-street, Gloucester-place.

1846 FREDERICK DAVIES, Surgeon to the Northern Dispensary; Upper Gower-street, Bedford-square.

1818 *HENRY DAVIES, M.D., 6, Duchess-street, Portland-place. C. 1827. V.P. 1848.

1847 JOHN DAVIES, M.D., Physician to the Hertford Infirmary, and Visiting Physician to the County Gaol and Lunatic Asylum, Hertford.

1853 ROBERT COKE NASH DAVIES, Winchelsea, Sussex.

1852 WILLIAM DAVIES, M.D., Senior Physician to the United Hospital, Bath; Gay-street, Bath.

1852 JOHN HALL DAVIS, M.D., Russell-place, Fitzroy-square.

1820 THOMAS DAVIS, Spring-gardens. C. 1843.

1818 JAMES DAWSON, Liverpool.

1847 GEORGE EDWARD DAY, M.D. F.R.S., Chandos Professor of Medicine, St. Andrew's.

Elected

1841 CAMPBELL DE MORGAN, Surgeon to, and Lecturer on Physiology at, the Middlesex Hospital; Upper Seymour-street, Portman-square. S. 1851-2.

1846 *SAMUEL BEST DENTON, Ivy-lodge, Hornsea, East Riding, Yorkshire.

1844 ROBERT DICKSON, M.D., Hertford-street, May-fair.

1839 JAMES DIXON, *Librarian;* Surgeon to the Royal London Ophthalmic Hospital; Green-street, Park-lane.

1845 JOHN DODD, 6, Upper Seymour-street, Portman-square.

1853 ROBERT DRUITT, M.D., Curzon-street, May-fair.

1846 JOHN DRUMMOND, Deputy-Inspector of Fleets and Hospitals; Royal Naval Hospital, Chatham.

1843 THOMAS JONES DRURY, M.D., Physician to the Salop Infirmary; Shrewsbury.

1845 GEORGE DUFF, M.D., Prospect-lodge, Elgin.

1845 EDWARD WILLSON DUFFIN, Langham-place, Portland-place.

1833 ROBERT DUNN, Norfolk-street, Strand. C. 1845.

1843 CHRISTOPHER MERCER DURRANT, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 HENRY SUMNER DYER, M.D., Bryanston-square. C. 1854.

1836 JAMES WILLIAM EARLE, Norwich.

1853 *GEORGE EDWARDES, Wolverhampton.

1824 GEORGE EDWARDS.

1823 CHARLES CHANDLER EGERTON, Kendall-lodge, Epping.

1848 GEORGE VINER ELLIS, Professor of Anatomy in University College, London.

1854 *JAMES ELLISON, M.D., Windsor.

1835 WILLIAM ENGLAND, M.D., Wisbeach, Cambridgeshire.

1842 JOHN ERIC ERICHSEN, Professor of Surgery in University College, London, and Surgeon to University College Hospital; Welbeck-street, Cavendish-square.

1815 *GRIFFITH FRANCIS DORSETT EVANS, M.D., High-street, Bedford. C. 1838.

1836 GEORGE FABIAN EVANS, M.D., Physician to the General Hospital, Birmingham.

1845 WILLIAM JULIAN EVANS, M.D.

1841 SIR JAMES EYRE, M.D., Consulting Physician to St. George's and St. James's Dispensary; Brook-street, Grosvenor-square. C. 1851.

Elected

1844 ARTHUR FARRE, M.D. F.R.S., Professor of Midwifery in King's College, London; Hertford-street, May-fair.

1831 ROBERT FERGUSON, M.D., Physician-Accoucheur to the Queen, Physician to the Westminster Lying-in Hospital; Park-street, Grosvenor-square. C. 1839. V.P. 1847.

1841 WILLIAM FERGUSSON, F.R.S., Professor of Surgery in King's College, London; Surgeon to King's College Hospital, and to H.R.H. Prince Albert; George-street, Hanover-square. C. 1849.

1852 ALFRED GEORGE FIELD, 46, Great Marlborough-street.

1850 *FREDERICK FIELD, Birmingham.

1849 GEORGE TUPMAN FINCHAM, M.D., Assistant-Physician to, and Lecturer on Forensic Medicine at, the Westminster Hospital, and Physician to the Western Dispensary; 28, Chapel-street, Belgrave square.

1836 JOHN WILLIAM FISHER, Surgeon-in-Chief to the Metropolitan Police Force; Grosvenor-gate. C. 1843.

1838 GEORGE LIONEL FITZMAURICE, Gloucester-place, Portman-square.

1842 THOMAS BELL ELCOCK FLETCHER, M.D., Physician to the General Hospital, Birmingham.

1841 SIR JOHN FORBES, M.D. F.R.S. D.C.L., Knt., Physician to her Majesty's Household; Old Burlington-street. C. 1852.

1848 JOHN GREGORY FORBES, Surgeon to the Western General Dispensary; Devonport-street, Hyde-park.

1852 JOHN COOPER FORSTER, Surgeon to the Surrey Dispensary; Wellington-street, Southwark.

1817 *ROBERT THOMAS FORSTER, Southwell, Notts.

1820 THOMAS FORSTER, M.D., Hartfield-lodge, East Grinstead.

1816 JOHN W. FRANCIS, M.D., Professor of Materia Medica in the University of New York, U.S.

1841 JOHN CHRISTOPHER AUGUST. FRANZ, M.D., Royal German Spa, Brighton.

1843 PATRICK FRASER, M.D., Physician to the London Hospital; Guildford-street, Russell-square.

1836 JOHN GEORGE FRENCH, Surgeon to St. James's Infirmary; Great Marlborough-street, Regent-street. C. 1852-3.

Elected

1849 ROBERT TEMPLE FRERE, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Queen-street, May-fair.

1846 HENRY WILLIAM FULLER, M.D., Assistant-Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; Manchester-square.

1815 *GEORGE FREDERICK FURNIVAL, Egham, Surrey.

1854 ALFRED BARING GARROD, M.D., Professor of Materia Medica and Therapeutics, University College, and Physician to University College Hospital; 63, Harley-street, Cavendish-square.

1851 GEORGE GASKOIN, Cambridge-terrace, Hyde-park.

1819 JOHN SAMUEL GASKOIN, Clarges-street, Piccadilly. C. 1836.

1819 HENRY GAULTER.

1848 JOHN GAY, Finsbury-place, Finsbury-square.

1821 *RICHARD FRANCIS GEORGE, Surgeon to the Bath Hospital.

1854 BERNARD GILPIN, Belle Vue-house, Ulverstone, Lancashire.

1851 STEPHEN JENNINGS GOODFELLOW, M.D., Assistant-Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital; Russell-square.

1818 JAMES ALEXANDER GORDON, M.D. F.R.S., Burford-lodge, Box-hill. C. 1828. V.P. 1829.

1851 PETER YEAMES GOWLLAND, Finsbury-square.

1844 JOHN GRANTHAM, Crayford, Kent.

1850 HENRY GRAY, F.R.S., Surgeon to the St. George's and St. James's Dispensary; Wilton-street, Grosvenor-place.

1846 GEORGE THOMPSON GREAM, M.D., 2, Upper Brook-street, Grosvenor-square.

1816 JOSEPH HENRY GREEN, F.R.S., Consulting Surgeon to St. Thomas's Hospital; Hadley, Middlesex. C. 1820. V.P. 1830.

1843 ROBERT GREENHALGH, M.D., Surgeon-Accoucheur to the Royal General Dispensary, St. Pancras; 11, Upper Woburn-place, Russell-square.

1814 JOHN GROVE, M.D., Salisbury.

1852 JOHN GROVE, Wandsworth, Surrey.

1849 WILLIAM WITHEY GULL, M.D., Assistant-Physician to Guy's Hospital; Finsbury-square.

1837 JAMES MANBY GULLY, M.D., Holyrood-house, Great Malvern.

Elected

1819 Sir John Gunning, Knight, C.B., Inspector of Hospitals; Paris

1842 Charles William Gardiner Guthrie, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital, and to the Westminster Ophthalmic Hospital; Pall Mall East.

1854 Samuel Osborne Habershon, M.D., Demonstrator of Morbid Anatomy, and Curator of the Museum, at Guy's Hospital; Physician to the City Dispensary; 48, Finsbury-circus, Finsbury-square.

1849 Hammett Hailey, Newport Pagnell, Bucks.

1852 Robert James Hale, M.D., 17, Westbourne-terrace, Hyde-park.

1842 *George Hale, M.D.

1845 John Hall, M.D., Deputy-Inspector-General of Hospitals; Cape of Good Hope.

1848 Alexander Halley, M.D., Queen Anne-street, Cavendish-square.

1819 Thomas Hammerton, Piccadilly. C. 1829.

1838 Henry Hancock, Surgeon to the Charing-cross Hospital; Harley-street, Cavendish-square. C. 1851.

1849 *Richard James Hansard, Broad-street, Oxford.

1848 *George Harcourt, M.D., Chertsey, Surrey.

1836 John Fosse Harding, Mylne-street, Myddleton-square.

1843 Thomas Sunderland Harrison, M.D. F.L.S., Garston-lodge, Somersetshire.

1846 John Harrison, the Court-yard, Albany.

1841 William Harvey, Surgeon to the Royal Dispensary for Diseases of the Ear, and to the Freemasons' Female Charity; Soho-square. C. 1854.

1853 Arthur Hill Hassall, M.D., Physician to the Royal Free Hospital; 8, Bennett-street, St. James's.

1828 Cæsar Henry Hawkins, President of the Royal College of Surgeons of England; Senior Surgeon to St. George's Hospital; Grosvenor-street, Grosvenor-square. C. 1830. V.P. 1838. T. 1841.

1838 Charles Hawkins, Savile-row, Regent-street. C. 1846. S. 1850.

1848 Thomas Hawksley, M.D., George-street, Hanover-square.

1820 Thomas Emerson Headlam, M.D., Newcastle-upon-Tyne.

Elected

1848 *James Newton Heale, M.D., Physician to the Winchester County Hospital; Winchester.

1850 George Heaton, M.D., Boston, U.S.

1829 Thomas Heberden, M.D., Park-street, Grosvenor-square.

1844 John Hennen, M.D., Physician to the Western General Dispensary; Upper Southwick-street, Hyde-park. L. 1848.

1848 Mitchell Henry, Assistant-Surgeon to the Middlesex Hospital; Harley-street, Cavendish-square.

1849 Amos Henriques, Upper Berkeley-street, Portman-square.

1821 Vincent Herberski, M.D., Professor of Medicine in the University of Wilna.

1843 Prescott Gardner Hewett, Assistant-Surgeon to the St. George's Hospital, Lecturer on Anatomy at St. George's Hospital Medical School; Hertford-street, May-fair.

1853 Thomas Hewlett, Surgeon to Harrow School; Harrow.

1841 *Nathaniel Highmore, Consulting-Surgeon to the Weymouth and Dorsetshire Eye Infirmary; Sherborne, Dorsetshire.

1814 *William Hill, Wootton-under-Edge, Gloucestershire.

1854 Thomas Hillier, B.A. (Lond.), Resident Medical Officer in University College Hospital.

1842 William Augustus Hillman, Assistant-Surgeon to, and Lecturer on Anatomy and Physiology at, Westminster Hospital; Argyll-street, Regent-street.

1841 John Hilton, F.R.S., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; New Broad-street, City. C. 1851.

1848 Martin Thomas Hiscox, M.D., Bath, Somersetshire.

1840 Thomas Hodgkin, M.D., Bedford-square. C. 1842.

1813 Joseph Hodgson, F.R.S., Westbourne-terrace, Hyde-park. C. 1817. P. 1851.

1835 Thomas Henry Holberton, Hampton, Middlesex.

1843 Luther Holden, 54, Gower-street, Bedford-square.

1814 Sir Henry Holland. Bart., M.D. F.R.S., Physician to the Queen, and Physician in Ordinary to H.R.H. Prince Albert; Brook-street, Grosvenor-square. C. 1817. V.P. 1826.

1846 Barnard Wight Holt, Surgeon to the Westminster Hospital; Parliament-street, Westminster.

Elected

1846 CARSTEN H. HOLTHOUSE, Surgeon to the Public Dispensary, Lincoln's Inn; Assistant-Surgeon to, and Lecturer on Anatomy and Physiology at, the Westminster Hospital; 9, New Burlington-street.

1853 WILLIAM CHARLES HOOD, M.D., Medical Superintendent, Bethlem Hospital.

1819 *JOHN HOWELL, M.D. F.R.S.E., Deputy-Inspector-General of Military Hospitals; Honorary and Consulting Physician to the Bristol Royal Infirmary; Datchet, near Windsor.

1828 *EDWARD HOWELL, M.D., Swansea, Glamorganshire.

1844 EDWIN HUMBY, Windsor-terrace, Maida-hill.

1822 ROBERT HUME, M.D. C.B., Inspector of Hospitals; Commissioner in Lunacy; Curzon-street, May-fair. V.P. 1836.

1840 HENRY HUNT, M.D., Brook-street, Hanover-square.

1842 CHRISTOPHER HUNTER, Downham, Norfolk.

1849 EDWARD LAW HUSSEY, Surgeon to the Radcliffe Infirmary, Oxford.

1820 WILLIAM HUTCHINSON, M.D.

1840 CHARLES HUTTON, M.D., Physician to the Royal Infirmary for Children; Assistant-Physician to the General Lying-in Hospital; Lowndes-street, Belgrave-square.

1838 WILLIAM IFIL, M.D.

1847 WILLIAM EDMUND IMAGE, Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk.

1826 WILLIAM INGRAM, Midhurst, Sussex.

1839 ALEXANDER RUSSELL JACKSON, M.D., Warley Barracks, Essex.

1845 *HENRY JACKSON, Surgeon to the Sheffield General Infirmary; St. James's-row, Sheffield.

1841 PAUL JACKSON, Bentinck-street, Manchester-square.

1847 THOMAS REYNOLDS JACKSON, Charles-street, St. James's.

1841 MAXIMILIAN MORITY JACOBOVICZ, M.D., Pesth.

1825 JOHN B. JAMES, M.D.

1847 *WILLIAM WITHALL JAMES, Exeter, Devonshire.

1844 SAMUEL JOHN JEAFFRESON, M.D., Leamington, Warwickshire.

1839 JULIUS JEFFREYS, F.R.S., Bath, Somersetshire.

Elected

1840 *George Samuel Jenks, M.D., Physician to the Sussex County Hospital; Brighton.

1851 William Jenner, M.D., Professor of Pathological Anatomy in University College, and Assistant-Physician to University College Hospital; Harley-street, Cavendish-square.

1848 Athol Archibald Wood Johnson, Lecturer on Physiology at St. George's Hospital Medical School, and Surgeon to the Hospital for Sick Children; 37, Albemarle-street.

1851 Edmund Charles Johnson, M.D., Savile-row, and Arlington-street, Piccadilly.

1821 Sir Edward Johnson, M.D., Weymouth, Dorsetshire.

1847 George Johnson, M.D., Assistant-Physician to King's College Hospital; Woburn-square.

1837 Henry Charles Johnson, Surgeon to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; Savile-row, Regent-street. C. 1850.

1844 John Johnston, Old Burlington-street.

1853 Henry Jones, Soho-square.

1844 Henry Bence Jones, M.D. F.R.S., Physician to St. George's Hospital; Grosvenor-street, Grosvenor-square.

1835 Henry Derviche Jones, Soho-square. C. 1854.

1853 Thomas Wharton Jones, F.R.S., 35, George-street, Hanover-square.

1837 Thomas William Jones, M.D., Physician to the City Dispensary; Finsbury-pavement, Finsbury-square.

1829 *George Charles Julius, Richmond, Surrey.

1816 *George Hermann Kauffmann, M.D., Hanover.

1815 Robert Keate, Serjeant-Surgeon to the Queen, Surgeon to H.R.H. the Duchess of Gloucester; Hertford-street, May-fair. C. 1818. V.P. 1826.

1848 *Daniel Burton Kendell, M.D., St. John's, Wakefield, Yorkshire.

1847 Alfred Keyser, Norfolk-crescent, Oxford-square.

1839 *David King, M.D., Eltham, Kent.

1851 John Abernethy Kingdon, New Bank-buildings, City.

1840 Samuel Armstrong Lane, Lecturer on Anatomy; Surgeon to the Lock Hospital, and to St. Mary's Hospital; Grosvenor-place, Hyde-park. C. 1849.

Elected

1841 *Charles Lashmar, M.D., Croydon, Surrey.

1816 G. E. Lawrence.

1809 William Lawrence, F.R.S., Surgeon Extraordinary to the Queen; Surgeon to St. Bartholomew's Hospital, and to Bridewell and Bethlem Hospital; Lecturer on Surgery at St. Bartholomew's Hospital; Whitehall-place, Whitehall. S. 1813. V.P. 1818. C. 1820. T. 1821. P. 1831.

1840 Thomas Laycock, M.D., York.

1843 *Jesse Leach, Heywood, near Bury, Lancashire.

1823 John G. Leath, M.D.

1822 John Joseph Ledsam, M.D.

1822 Robert Lee, M.D. F.R.S., Physician to the British Lying-in Hospital; Physician-Accoucheur to the St. Marylebone Infirmary; and Lecturer on Midwifery at St. George's Hospital; Savile-row, Regent-street. C. 1829. S. 1830. V.P. 1835.

1843 Henry Lee, M.D., Keppel-street, Russell-square. C. 1837. S. 1839.

1843 Henry Lee, Assistant-Surgeon to King's College Hospital, and Surgeon to the Lock Hospital; Dover-street, Piccadilly.

1851 George Macartney Leese, Gloucester-place, Portman-square.

1836 Frederick Leighton, M.D., Frankfort-on-the-Maine.

1854 Hananel de Leon, M.D., 4, Gordon-street, Gordon-square.

1847 John Charles Weaver Lever, M.D., Physician-Accoucheur to Guy's Hospital; Wellington-street, Southwark.

1847 Sir John Liddell, M.D. F.R.S. C.B., Inspector of Hospitals; Royal Hospital, Greenwich.

1806 John Lind, M.D.

1845 William John Little, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 34, Brook-street, Grosvenor-square.

1819 Robert Lloyd, M.D.

1824 Eusebius Arthur Lloyd, Surgeon to St. Bartholomew's and Christ's Hospitals; Bedford-row. S. 1827. V.P. 1838. C. 1843.

Elected

1820 J. G. Locher, M.C.D., Town Physician of Zurich.

1844 Edward Francis Lonsdale, Surgeon to the Royal Orthopædic Hospital; Montague-street, Russell-square.

1824 Charles Locock, M.D., First Physician-Accoucheur to the Queen, and Consulting Physician to the General Lying-in Hospital; Hertford-street, May-fair. C. 1826. V.P. 1841.

1852 Charles Lodge, M.D.

1846 Henry Thomas Lomax, Stafford.

1836 Joseph S. Löwenfeld, M.D., Berbice.

1815 *Peter Luard, M.D.

1852 James Luke, Senior-Surgeon to the London Hospital; Vice-President of the Royal College of Surgeons of England; Broad-street-buildings.

1846 William M'Ewen, M.D., Surgeon to the Cheshire County Gaol, and House-Surgeon to the Chester General Infirmary; Newgate-street, Chester.

1814 Sir James Macgrigor, Bart., M.D. K.C.B. K.T.S. LL.D. F.R.S. L. and E., Director-General of the Medical Department of the Army; Harley-street, Cavendish-square. C. 1820. V.P. 1815.

1823 George Macilwain, Consulting Surgeon to the Finsbury Dispensary; the Court-yard, Albany. C. 1829. V.P. 1848.

1839 William Macintyre, M.D., Harley-street, Cavendish-square. C. 1850.

1848 Frederick William Mackenzie, M.D., Chester-place, Hyde-park-square.

1818 William Mackenzie, Surgeon to the Eye Infirmary, Glasgow.

1854 *Draper Mackinder, M.D., Gainsborough, Lincolnshire.

1822 Richard Mackintosh, M.D.

1844 Daniel Maclachlan, M.D., Physician to the Royal Hospital, Chelsea, and Deputy-Inspector-General of Hospitals; Royal Hospital, Chelsea.

1851 Samuel Maclean, Brook-street, Grosvenor-square.

1849 Duncan Maclachlan Maclure, Harley-street, Cavendish-square.

1842 John Macnaught, M.D., Bedford-street, Liverpool.

Elected

1835 DANIEL CHAMBERS MACREIGHT, M.D., St. Hillier's, Jersey.

1837 ANDREW MELVILLE M'WHINNIE, Assistant-Surgeon to St. Bartholomew's Hospital; Lecturer on Comparative Anatomy at St. Bartholomew's Hospital; Assistant-Surgeon to the London Hospital for Diseases of the Skin; Bridge-street, Blackfriars. C. 1851.

1848 WILLIAM ORLANDO MARKHAM, M.D., Assistant-Physician to St. Mary's Hospital; Clarges-street, Piccadilly.

1824 SIR HENRY MARSH, Bart., M.D., Dublin.

1838 THOMAS PARR MARSH, M.D., Physician to the Salop Infirmary, Shrewsbury.

1851 JOHN MARSHALL, Assistant-Surgeon to University College Hospital; 10, George-street, Hanover-square.

1841 JAMES RANALD MARTIN, F.R.S., Lower Grosvenor-street, Grosvenor-square. C. 1853.

1849 GEORGE BELLASIS MASFEN, 78, Oxford-street, Manchester.

1853 WILLIAM EDWARD MASFEN, Stafford.

1818 J. P. MAUNOIR, Professor of Surgery at Geneva.

1837 THOMAS MAYO, M.D. F.R.S., Physician to the St. Marylebone Infirmary; Wimpole-street, Cavendish-square. S. 1841. C. 1847. V.P. 1851.

1839 RICHARD HENRY MEADE, Bradford, Yorkshire.

1819 *THOMAS MEDHURST, Hurstbourne Tarrant, Hampshire.

1837 SAMUEL WILLIAM JOHN MERRIMAN, M.D., Physician to the Royal Infirmary for Children; Consulting Physician to the Westminster General Dispensary; and Assistant-Physician to the West London Lying-in Institution; 3, Charles-street, Westbourne-terrace, Hyde-park.

1852 JAMES MERRYWEATHER, 57, Brook-street, Grosvenor-square.

1847 EDWARD MERYON, M.D., Clarges-street, Piccadilly.

1815 AUGUSTUS MEYER, M.D., St. Petersburgh.

1840 RICHARD MIDDLEMORE, Consulting-Surgeon to the Eye Infirmary, Birmingham.

1854 EDWARD ARCHIBALD MIDDLESHIP, Richmond, Surrey.

1818 *PATRICK MILLER, M.D. F.R.S. E., Physician to the Devon and Exeter Hospitals, and to the Lunatic Asylum; Exeter, Devonshire.

1848 GAVIN MILROY, M.D., 55, Victoria-street, Westminster.

Elected

1852 JAMES MONRO, M.D., Surgeon-Major, Coldstream Guards; Vincent-square, Westminster.

1844 NATHANIEL MONTEFIORE, 4, Stanhope-street, May-fair.

1828 JOSEPH MOORE, M.D., *Treasurer;* Physician to the Royal Freemasons' Female Charity; Consulting Physician to Queen Charlotte's Lying-in Hospital; Savile-row, Regent-street. C. 1837.

1836 GEORGE MOORE, M.D., Hastings.

1848 CHARLES HEWITT MOORE, Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 35, Montague-place, Russell-square.

1854 GEORGE MOSELEY, Sandgate, Kent.

1851 FREDERICK JOHN MOUAT, M.D., Professor of Medicine in the Medical College of Calcutta, and Secretary of the Council of Education in India; Calcutta.

1814 *GEORGE FREDERICK MUHRY, M.D., Hanover.

1847 SIMON MURCHISON, Steepleaston, near Woodstock, Oxon.

1845 THOMAS D. MUTTER, M.D., Professor of Surgery in Jefferson Medical College; Philadelphia.

1840 ROBERT NAIRNE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Charles-street, Berkeley-square. C. 1848.

1835 THOMAS ANDREW NELSON, M.D., Nottingham-terrace, New-road.

1843 EDWARD NEWTON, Howland-street, Fitzroy-square.

1851 JAMES NICHOLS, Savile-row, Regent-street.

1819 *GEORGE NORMAN, Surgeon to the United Hospital and Puerperal Charity; Bath.

1849 HENRY BURFORD NORMAN, Surgeon to the North London Eye Infirmary, and the St. Marylebone Dispensary; Duchess-street, Portland-place.

1845 HENRY NORRIS, South Petherton, Somerset.

1849 *ARTHUR NOVERRE, Great Stanmore, Middlesex.

1847 *WILLIAM EDWARD CHARLES NOURSE.

1843 WILLIAM O'CONNOR, M.D., 30, Upper Montague-street, Montague-square.

1847 THOMAS O'CONNOR, March, Cambridgeshire.

1846 FRANCIS ODLING, Devonshire-street, Portland-place.

Elected

1822 JAMES ADEY OGLE, M.D. F.R.S., Clinical and Aldrichian Professor of Medicine, Oxford; and Senior Physician to the Radcliffe Infirmary; Oxford.

1850 HENRY OLDHAM, M.D., Obstetric Physician to Guy's Hospital; Finsbury-square.

1842 WILLIAM PIERS ORMEROD.

1846 *EDWARD LATHAM ORMEROD, M.D., Physician to the Sussex County Hospital; Old Steyne, Brighton.

1847 WILLIAM EMANUEL PAGE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Curzon-street, May-fair.

1847 *WILLIAM BOUSFIELD PAGE, Surgeon to the Cumberland Infirmary; Carlisle.

1840 JAMES PAGET, F.R.S., Assistant-Surgeon to, and Lecturer on General and Morbid Anatomy and Physiology at, St. Bartholomew's Hospital; Henrietta-street, Cavendish-square. C. 1848.

1806 *ROBERT PALEY, M.D., Bishopston-grange, near Ripon, Yorkshire.

1836 S. W. LANGSTON PARKER, Surgeon to the Queen's Hospital; Birmingham.

1847 NICHOLAS PARKER, M.B., Assistant-Physician to the London Hospital: Microscopical Demonstrator of Morbid Anatomy at the London Hospital School of Medicine; Finsbury-square.

1841 JOHN PARKIN, M.D., Paris.

1851 JAMES PART, 7, Camden-road-villas, Camden-town.

1828 RICHARD PARTRIDGE, F.R.S., Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London; New-street, Spring-gardens. S. 1832. C. 1837. V.P. 1847.

1845 THOMAS BEVILL PEACOCK, M.D., Assistant-Physician to St. Thomas's Hospital; Finsbury-circus, Finsbury-square,

1830 CHARLES P. PELECHIN, M.D., St. Petersburgh.

1819 JOHN PRYOR PEREGRINE, M.D., Jersey.

1839 THOMAS PEREGRINE, M.D., Half Moon-street, Piccadilly.

1844 WILLIAM VESALIUS PETTIGREW, M.D., Chester-street, Grosvenor-place.

Elected

1837 Benjamin Phillips, F.R.S., Brentbridge-house, Hendon, Middlesex. L. 1841. T. 1847.

1814 *Edward Phillips, M.D., Consulting Physician to the County Hospital; Winchester, Hampshire.

1848 Edward Phillips, M.D., Coventry, Warwickshire.

1852 Richard Phillips, 5, Winchester-place, Pentonville.

1854 Thomas Bacon Phillips, 24, Powis-square, Brighton.

1846 Francis Richard Philp, M.D., Colby-house, Kensington.

1851 *James Hollins Pickford, M.D. M.R.I.A., Brighton.

1851 John Picton, M.D.

1836 Isaac Pidduck, M.D., Physician to the Bloomsbury Dispensary; Montague-street, Russell-square.

1852 George Pilcher, Harley-street, Cavendish-square.

1852 Henry Pilleau, 11, Young-street, Kensington-square.

1841 Henry Alfred Pitman, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Montague-place, Russell-square.

1850 Alfred Poland, Assistant-Surgeon to Guy's Hospital, and to the Royal Ophthalmic Hospital; St. Helen's-place, Bishopsgate-street.

1845 George David Pollock, Surgeon to the North London Eye Infirmary; Assistant-Surgeon to, and Lecturer on Anatomy at, St. George's Hospital Medical School; Grosvenor-street, Grosvenor-square.

1843 Charles Pope, M.D. M.A. F.L.S., Temple Cloud, near Bristol.

1846 Jephson Potter, M.D. F.L.S. M.R.C.S., Oxford-road, Manchester.

1840 Lewis Powell, John-street, Berkeley-square.

1842 James Powell, M.B. (Lond.), Guildford-street, Russell-sq.

1851 Robert Francis Power, M.D., 14, Waterloo-place.

1839 John Propert, New Cavendish-street, Portland-place.

1845 John Pyle, Surgeon to the North London Eye Infirmary; Oxford-terrace, Hyde-park.

1816 Sir William Pym, M.D., Inspector of Hospitals.

1830 Jones Quain, M.D., Paris.

1850 Richard Quain, M.D., Assistant-Physician to the Hospital for Consumption; Harley-street, Cavendish-square.

Elected

1835 RICHARD QUAIN, F.R.S., Surgeon to University College Hospital, and Professor of Clinical Surgery in University College, London; Cavendish-square. C. 1838. L. 1846. T. 1851.

1852 CHARLES BLAND RADCLIFFE, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; Henrietta-street, Cavendish-square.

1854 *WILLIAM HENRY RANSOM, M.D., Nottingham.

1821 HENRY REEDER, M.D., Ridge-house, Chipping, Sudbury.

1835 G. REGNOLI, Professor of Surgery in the University of Pisa.

1846 JAMES REID, M.D., Physician to the Infirmary of St. Giles and Bloomsbury; General Lying-in Hospital, &c.; Brook-street, Grosvenor-square.

1847 SAMUEL RICHARDS, M.D., Bedford-square.

1853 CHRISTOPHER THOMAS RICHARDSON, M.B., 16, Hinde-street, Manchester-square.

1829 SIR JOHN RICHARDSON, Knt., F.R.S. C.B., Surgeon to the Naval Hospital; Haslar Hospital, Gosport.

1849 *WILLIAM RICHARDSON, M.D., 9, Ephraim-road, Tunbridge Wells, Kent.

1843 JOSEPH RIDGE, M.D., Dorset-square.

1845 BENJAMIN RIDGE, M.D., Putney, Surrey.

1852 CHARLES RIDLEY, Charlotte-street, Bedford-square.

1852 JOHN ROBERTS, M.D., Physician to the Westminster General Dispensary; Bruton-street, Berkeley-square.

1829 *ARCHIBALD ROBERTSON, M.D. F.R.S. L. and E., Physician to the General Infirmary, Northampton.

1843 GEORGE ROBINSON, M.D., Newcastle-on-Tyne.

1843 WILLIAM RODEN, M.D. F.L.S., Kidderminster.

1835 GEORGE HAMILTON ROE, M.D., Physician to the Westminster Hospital; Upper Brook-street, Grosvenor-square. C. 1841.

1836 ARNOLD ROGERS, Hanover-square.

1846 WILLIAM RICHARD ROGERS, M.D., Berners-street, Oxford-street.

1819 HENRY SHUCKBURGH ROOTS, M.D., Consulting-Physician to St. Thomas's Hospital; Russell-square. C. 1833. V.P. 1834.

1829 *WILLIAM SUDLOW ROOTS, Kingston, Surrey.

Elected

1850 GEORGE ROPER, 180, Shoreditch.
1836 RICHARD ROSCOE, M.D., Twickenham, Middlesex.
1836 *CALEB BURRELL ROSE, Swaffham, Norfolk.
1850 ARCHIBALD COLQUHOUN ROSS, M.D., Madeira.
1849 CHARLES HENRY FELIX ROUTH, M.D., 52, Montague-square.
1845 HENRY MORTIMER ROWDON, 29, Nottingham-place, York-gate, Regent's-park.
1841 RICHARD ROWLAND, M.D., Assistant-Physician to the Charing-cross Hospital; Woburn-place, Russell-square.
1834 HENRY WILLIAM RUMSEY, Cheltenham.
1845 JAMES RUSSELL, M.D., Physician to the General Dispensary, Birmingham.
1851 HENRY HYDE SALTER, M.B., Montague-street, Russell-sq.
1827 *THOMAS SALTER, F.L.S., Poole, Dorsetshire.
1844 *THOMAS BELL SALTER, M.D. F.L.S., Ryde, Isle of Wight.
1849 HUGH JAMES SANDERSON, M.D., Upper Berkeley-street, Portman-square.
1847 WILLIAM HENRY OCTAVIUS SANKEY, M.D., London Fever Hospital, Liverpool-road, Islington.
1845 EDWIN SAUNDERS, Surgeon-Dentist to the Queen, and Lecturer on Diseases of the Teeth at St. Thomas's Hospital; George-street, Hanover-square.
1834 LUDWIG V. SAUVAN, M.D., Warsaw.
1840 AUGUSTIN SAYER, M.D., Upper Seymour-street, Portman-square.
1853 MAURICE SCHULHOF, M.D., Physician to the Royal General Dispensary, Bartholomew-close; Suffolk-place, Pall Mall.
1824 EDWARD JAMES SEYMOUR, M.D. F.R.S., Charles-street, Berkeley-square. C. 1826. S. 1827. V.P. 1830.
1840 WILLIAM SHARP, F.R.S. F.G.S. F.R.A.S., Rugby.
1837 WILLIAM SHARPEY, M.D. F.R.S. L. and E., Professor of Anatomy and Physiology in University College, London, and Secretary of the Royal Society; Gloucester-crescent, Regent's-park. C. 1848.
1836 ALEXANDER SHAW, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; Henrietta-street, Cavendish-square. C. 1842. S. 1843. V.P. 1851.
1848 *EDWARD JAMES SHEARMAN, M.D., Rotherham, Yorkshire.

Elected

1849 FRANCIS SIBSON, M.D. F.R.S., Physician to St. Mary's Hospital; Brook-street, Grosvenor-square.

1848 EDWARD HENRY SIEVEKING, M.D., Assistant-Physician to St. Mary's Hospital; Bentinck-street, Manchester-sq.

1839 THOMAS HOOKHAM SILVESTER, M.D., High-street, Clapham. C. 1854.

1842 JOHN SIMON, F.R.S., Surgeon and Lecturer on Pathology at St. Thomas's Hospital; Upper Grosvenor-street. C. 1854.

1821 CHARLES SKENE, M.D., Professor of Anatomy and Surgery; Marischal College, Aberdeen.

1827 GEORGE ROBERT SKENE, Bedford.

1824 FREDERIC CARPENTER SKEY, F.R.S., Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Northern Dispensary; Grosvenor-street, Grosvenor-square. C. 1828. L. 1829. V.P. 1841.

1838 HENRY SPENCER SMITH, Senior Assistant-Surgeon to St. Mary's Hospital; and Lecturer on Surgery in the Medical School adjoining St. George's Hospital; Sussex-gardens, Hyde-park. C. 1854.

1835 JOHN GREGORY SMITH, Harewood, Yorkshire.

1843 ROBERT WILLIAM SMITH, M.D. M.R.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; Dublin.

1852 CHARLES CASE SMITH, Senior-Surgeon to the Suffolk General Hospital; Bury St. Edmunds, Suffolk.

1845 WILLIAM SMITH, Park-street, Bristol.

1847 WILLIAM SMITH, M.D., Weymouth, Dorsetshire.

1850 WILLIAM TYLER SMITH, M.D., Physician-Accoucheur to St. Mary's Hospital; Upper Grosvenor-street, Grosvenor-square.

1843 JOHN SNOW, M.D., Sackville-street, Piccadilly.

1851 JOHN SODEN, Surgeon to the Bath Hospital; Bath.

1816 *JOHN SMITH SODEN, New Sidney-place, Bath.

1830 SAMUEL SOLLY, F.R.S., Surgeon to St. Thomas's Hospital; St. Helen's Place, Bishopsgate-street. L. 1838. C. 1845. V.P. 1849.

1844 FREDERICK ROBERT SPACKMAN, M.B., Harpenden, St. Alban's.

1834 JAMES SPARK, Newcastle, Staffordshire.

Elected

1851 ROBERT JOHN SPITTA, M.B., Clapham, Surrey.

1843 *STEPHEN SPRANGER, Grantham, Lincolnshire.

1838 GEORGE JAMES SQUIBB, 11, Montague-place, Montague-sq.

1815 EDWARD STANLEY, F.R.S., Surgeon to St. Bartholomew's Hospital; Brook-street, Grosvenor-square. C. 1821. S. 1824. V.P. 1827. T. 1832. P. 1843. C. 1852-3.

1851 JAMES STARTIN, Surgeon to the Hospital for Diseases of the Skin, and Lecturer on Cutaneous Disorders at that Institution; Savile-row, Regent-street.

1852 SHERARD FREEMAN STATHAM, Assistant-Surgeon to University College Hospital; 43, Mortimer-street, Cavendish-square.

1854 HENRY STEVENS, Resident Medical Officer, St. Luke's Hospital; St. Luke's.

1842 ALEXANDER PATRICK STEWART, M.D, Assistant-Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital; Grosvenor-street, Grosvenor-square.

1843 ROBERT REEVE STORKS.

1844 JOHN SOPER STREETER, Harpur-street, Red Lion-square.

1847 WILLIAM ALLEN SUMNER, Surgeon to the Portland Town Free Dispensary; 25, Wellington-toad, St. John's-wood,

1839 ALEXANDER JOHN SUTHERLAND, M.D. F.R.S., Physician to St. Luke's Hospital; Richmond-terrace, Whitehall. C. 1850.

1842 JAMES SYME, Professor of Clinical Surgery in the University of Edinburgh; Charlotte-square, Edinburgh.

1854 *FREDERICK SYMONDS, Surgeon to the Radcliffe Infirmary; 32, Beaumont-street, Oxford.

1844 RICHARD WILLIAM TAMPLIN, Surgeon to the Royal Orthopædic Hospital; Old Burlington-street.

1848 THOMAS HAWKES TANNER, M.D., Physician to the Hospital for Women, Soho-square; Charlotte-street, Bedford-square.

1840 THOMAS TATUM, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; George-street, Hanover-square. C. 1852-3.

1835 JOHN COLLEY TAUNTON, Surgeon to the City of London Truss Society, and to the City Dispensary; Hatton-garden, Holborn. C. 1840.

Elected

1845 Thomas Taylor, Vere-street, Cavendish-square.

1852 Robert Taylor, M.D., 82, Guildford-street, Russell-square.

1845 *Evan Thomas, Manchester.

1839 Seth Thompson, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; Lower Seymour-street, Portman-square. C. 1849. S. 1850.

1842 Theophilus Thompson, M.D. F.R.S., Physician to the Hospital for Consumption and Diseases of the Chest Bedford-square.

1852 Henry Thompson, Surgeon to the St. Marylebone Dispensary and Infirmary; 16, Wimpole-street, Cavendish-square.

1835 Frederick Hale Thomson, Clarges-street, Piccadilly.

1819 John Thomson, M.D. F.L.S., Physician to the Finsbury Dispensary; Dalby-terrace, Islington. C. 1833. S. 1834. V.P. 1850.

1850 Robert Dundas Thomson, M.D., Professor of Chemistry, University of Glasgow.

1836 John Thurnam, M.D., Devizes, Wiltshire.

1848 Edward John Tilt, M.D., Physician to the Farringdon Dispensary; York-street, Portman-square.

1834 Robert Bentley Todd, M.D. F.R.S., *Vice-President;* Physician to King's College Hospital, Professor of Physiology and of General and Morbid Anatomy in King's College, London; Brook-street, Grosvenor-square. L. 1842. T. 1850.

1828 James Torrie, M.D., Aberdeen.

1843 Joseph Toynbee, F.R.S., Aural Surgeon to St. Mary's Hospital, Consulting Aural Surgeon to the Asylum for the Deaf and Dumb, and Consulting Surgeon to the St. George's and St. James's General Dispensary; Savile-row, Regent-street.

1850 Samuel John Tracy, Surgeon-Dentist to St. Bartholomew's and Christ's Hospitals; Finsbury-place, Finsbury-square.

1808 Benjamin Travers, F.R.S., Surgeon Extraordinary to the Queen, Surgeon in Ordinary to His Royal Highness Prince Albert; Green-street, Grosvenor-square. C. 1810. V.P. 1817. P. 1827.

Elected

1841 MATTHEW TRUMAN, M.D., Norland-square, Notting-hill.

1835 JOHN CUSSON TURNER, M.D., Brighton.

1845 THOMAS TURNER, Surgeon to the Royal Manchester Infirmary, and Lecturer on Anatomy; Mosley-st., Manchester.

1846 ALEXANDER URE, Surgeon to St. Mary's Hospital, and Consulting Surgeon to the Westminster General Dispensary; 18, Upper Seymour-street, Portman-square.

1819 BARNARD VAN OVEN, M.D., Consulting Surgeon to the Charity for Delivering Jewish Lying-in Women; 22, Manchester-square.

1806 BOYER VAUX, M.D.

1839 WILLIAM RANDALL VICKERS, Baker-street, Portman-square.

1810 JAMES VOSE.

1828 BENEDETTO VULPES, M.D., Physician to the Hospital of Aversa, and to the Hospital of Incurables, Naples.

1854 EDWARD WADDINGTON, 2, Guildford-place, Russell-square.

1841 ROBERT WADE, Surgeon to the Westminster General Dispensary; Dean-street, Soho.

1823 WILLIAM WAGNER, M.D., Berlin.

1820 THOMAS WALKER, M.D., Physician to the Forces; Morro Velhio, Brasil.

1852 WALTER HAYLE WALSHE, M.D., Professor of the Theory and Practice of Medicine in University College, and Physician to University College Hospital; 40, Queen Anne-street, Cavendish-square.

1851 HENRY HAYNES WALTON, Surgeon to the Central London Ophthalmic Hospital, and Assistant-Surgeon to St. Mary's Hospital; Brook-street, Hanover-square.

1852 DANIEL WANE, M.D., 20, Grafton-street, Berkeley-square.

1846 NATHANIEL WARD, Assistant-Surgeon to, and Demonstrator of Anatomy at, the London Hospital; Broad-street-buildings, City.

1845 THOMAS OGIER WARD, M.D., Leonard-place, Kensington.

1821 WILLIAM TILLEARD WARD, Duncannon-house, Brighton.

1846 JAMES THOMAS WARE, Surgeon to the Finsbury Dispensary, and to the Convalescent Institution; Russell-square.

1811 JOHN WARE, Clifton, near Bristol.

1814 MARTIN WARE, Russell-square, *Vice-President*. C. 1844. T. 1846.

Elected

1816 *Charles Bruce Warner, Cirencester, Gloucestershire.

1829 Elias Taylor Warry, Wimborne, Dorsetshire.

1837 Thomas Watson, M.D., Henrietta-street, Cavendish-square. C. 1840. V.P. 1845. C. 1852.

1847 *Thomas Watson, Holbeach, Lincolnshire.

1854 William Webb, M.D., Resident Medical Officer of the Stafford General Infirmary; Stafford.

1840 William Woodham Webb, Gislingham, near Thwaite, Suffolk.

1842 Frederick Weber, M.D., Physician to the St. George's and St. James's Dispensary; Green-street, Park-lane.

1835 John Webster, M.D. F.R.S., Consulting Physician to the St. George's and St. James's Dispensary; Brook-street, Grosvenor-square. C. 1843.

1844 William Wegg, M.D., *Librarian*; Physician to the St. George's and St. James's Dispensary; Maddox-street, Hanover-square.

1854 Thomas Spencer Wells, 30, Brook-street, Grosvenor-square.

1816 Sir Augustus West, Knt., Deputy-Inspector of Hospitals to the Portuguese Forces; Paris.

1842 Charles West, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital; and Physician to the Hospital for Sick Children; Wimpole-street, Cavendish-square.

1841 Thomas West, M.D. F.L.S., Daventry.

1828 John Whatley, M.D.

1849 John White, the Albany, Piccadilly.

1852 John Wiblin, 73, Morland-place, Southampton.

1840 Joseph Wickenden, Birmingham.

1824 *William John Wickham, Surgeon to the Winchester Hospital; Winchester.

1844 Frederick Wildbore, 1, Trafalgar-place East, Hackney-road.

1837 George Augustus Frederick Wilks, M.D., Temple-walk, Matlock, Derbyshire.

1840 Charles James Blasius Williams, M.D. F.R.S., Upper Brook-street, Grosvenor-square. C. 1849.

1829 Robert Willis, M.D., Barnes, Surrey. L. 1838.

Elected

1839 ERASMUS WILSON, F.R.S., Consulting Surgeon to the St. Pancras Infirmary; Henrietta-street, Cavendish-square.

1839 JAMES ARTHUR WILSON, M.D., Physician to St. George's Hospital; Dover-street, Piccadilly. C. 1846.

1831 WILLIAM JAMES WILSON, Surgeon to the Manchester Infirmary; Manchester.

1850 *ROBERT STANTON WISE, M.D., Banbury, Oxon.

1825 THOMAS ALEXANDER WISE, India.

1851 JOHN WOOD, 21, Newcastle-street, Strand.

1841 GEORGE LEIGHTON WOOD, Surgeon to the Bath Hospital; Queen-square, Bath.

1848 WILLIAM WOOD, M.D., Kensington-house, Kensington.

1843 JOHN WARD WOODFALL, M.D., Physician to the West Kent Infirmary; Maidstone, Kent.

1833 THOMAS WORMALD, *Vice-President;* Assistant-Surgeon to St. Bartholomew's Hospital; Bedford-row. C. 1839.

1842 WILLIAM COLLINS WORTHINGTON, Surgeon to the Infirmary, Lowestoft, Suffolk.

1848 EDWARD JOHN WRIGHT, Kennington-row, Kennington.

[It is particularly requested, that any change of Title or Residence may be communicated to the Secretaries before the 1st of August in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1841 WILLIAM THOMAS BRANDE, F.R.S.L. and E., Professor of Chemistry at the Royal Institution of Great Britain; Royal Mint, Tower-hill.

1835 SIR DAVID BREWSTER, K.H. LL.D F.R.S. L. and E., &c., Cor. Mem. Institute of France, &c.; Kingussie.

1853 BENJAMIN COLLINS BRODIE, B.A., Oxford, F.R.S.; 13, Albert-road, Regent's-park.

1841 ROBERT BROWN, D.C.L. F.R.S., President of the Linnean Society; British Museum.

1835 The Very Rev. WILLIAM BUCKLAND, D.D. F.R.S., Dean of Westminster.

1847 EDWIN CHADWICK, Commissioner of the Board of Health.

1835 MICHAEL FARADAY, D.C.L. F.R.S., Cor. Memb. Institute of France; Royal Institution.

1841 SIR JOHN FREDERICK WILLIAM HERSCHEL, Bart., D.C.L. F.R.S., President of the Royal Astronomical Society; Somerset House.

1835 SIR WILLIAM JACKSON HOOKER, LL.D. F.R.S. L. and E., Royal Botanic Garden, Kew.

1847 RICHARD OWEN. F.R.S., Cor. Memb. Institute of France; Hunterian Professor to, and Curator of the Museum of, the Royal College of Surgeons of England.

1835 The Rev. ADAM SEDGWICK, A.M. F.R.S., &c., Woodwardian Lecturer, Cambridge.

1841 The Rev. WILLIAM WHEWELL, D.D. F.R.S., Master of Trinity College, Cambridge.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1841 G. ANDRAL, M.D., Professor in the Faculty of Medicine, Paris.

1835 CARL JOHAN ECKSTRÖM, K.P.S. and W., Physician to the King of Sweden, First Surgeon to the Seraphim Hospital, Stockholm.

1841 CHRISTIAN GOTTFRIED EHRENBERG, Berlin.

1835 BARON A. DE HUMBOLDT, Member of the Institute of France, &c., Berlin.

1841 JAMES JACKSON, M.D., Professor of Medicine in the University of Cambridge, Boston, U.S.

1843 BARON JUSTUS LIEBIG, M.D. F.R.S., Professor of Chemistry in the University of Giessen, &c.

1841 P. C. A. LOUIS, M.D., Physician to the Hôtel-Dieu, Member of the Royal Academy of Medicine, &c., Paris.

1841 F. MAGENDIE, M.D., Member of the Institute; Physician to the Hospital of the Salpêtrière; Paris.

1847 PROFESSOR CARLO MATTEUCCI, University of Pisa.

1841 JOHANN MULLER, M.D., Professor of Anatomy and Physiology, and Director of the Royal Anatomical Museum, Berlin.

1841 BARTOLOMEO PANIZZA, M.D., Pavia.

1850 CARL ROKITANSKY, M.D., Curator of the Imperial Pathological Museum at the University of Vienna, &c. &c.

1853 VALENTINE MOTT, M.D., New-York.

1835 C. J. TIMMINCK, Director of the Museum of Natural History of the King of Holland, Amsterdam.

1835 FREDERICK TIEDEMANN, M.D., Professor of Anatomy and Physiology, Heidelberg.

1841 JOHN C. WARREN, M.D., Professor of Anatomy and Surgery in the University of Cambridge, Boston, U.S.

SCROFULOUS CARIES

OF

THE LEFT ASTRAGALUS.

EXCISION—CURE,

WITH FORMATION OF A FRESH JOINT.

BY

S. F. STATHAM,

ASSISTANT-SURGEON, UNIVERSITY COLLEGE HOSPITAL.

Received Nov. 7th, 1853.—Read Jan. 24th, 1854.

Henry Cudden, æt. 5, of strumous tendency, was said to have had weakness of the left ankle since birth. At Christmas, 1851, a swelling appeared below the outer side of the left ankle, which was blistered; since May he has been under hospital treatment; painting with iodine was frequently employed; latterly the formation of matter pointed naturally on the inner side, and required opening outside the joint.

August 25, 1852.—The integument was much diseased about the ankle; but on closer examination, and after a week's rest in the hospital, it was found to be actually implicated only where corresponding to the situation of the astragalus. Chloroform being administered, the fistulæ were thoroughly examined. On the outer side the probe reached the surface

of the astragalus, which was exposed and softened, and there was a fistula leading backwards by the side of the calcaneum; on the inner side a probe passed easily along the posterior face of the astragalus. The ankle-joint was healthy.

Medicines and local applications having been fairly tried without avail, amputation below the knee would probably become inevitable; and as his health was already materially suffering, resection of the astragalus, and of any portion of the calcaneum that might be necessary was considered justifiable, if only to be followed subsequently by removal of the limb.

August 27.—My friends Messrs. Marshall and Glover assisting me, an incision, three inches long, was carried along the outer side of the extensor tendons of the toes, and another to fall into the middle of this one from the outer side of the foot. The finger found carious disease of the neighbouring surfaces of the astragalus and calcaneum. Having lifted up the flaps of the soft parts, and separated the tendons and vessels in front of the joint in one mass from the bone, it was sought to release its head, which proving troublesome, all difficulty was at once removed by cutting through the neck of the astragalus with the scalpel, and then by means of the fingers and sequestrum-forceps, the pieces were dragged out, while the knife freed them from the surrounding parts. During extraction, the posterior portion of the upper cartilaginous surface became separated from the body of the bone, and was removed later; this circumstance much facilitated the operation. The upper surface of the calcaneum, for its posterior two thirds, was found to be carious, and was therefore gouged off to a depth of about one eighth of an inch. The foot hung perfectly loose; three fingers could be easily introduced to the bottom of the wound, the surfaces of the tibia and fibula were sound, the remaining portions of the tarsus offered no reasons for interfering with them. It was found that the tendons of the peroneus brevis and external tendon of the extensor of the toes had been divided, the lateral ligaments to the calcaneum had escaped, and no vessels required ligature, the profuse hæmorrhage

being readily checked by cold water. Lint was introduced into the cavity of the wound. The same evening a splint was applied on the inner side of the leg and foot, and a piece of wet lint laid over the wound; this was still large and gaping, as the calcaneum would not enter between the malleoli. The splint and pad was perforated to allow the escape of any wound-secretions.

On examination of the bone, the upper articular cartilage appeared to be unaffected, but easily separated from the carious body of the bone beneath. The posterior articulation of the astragalus with the calcaneum had disappeared. The head and neck of the bone appeared to be sound.

September 1st.—Wound suppurating, health fair. Was put on iron and nitric acid, later porter; fish, &c. The foot was never removed from the splint, nor the watery pus from the cavity of the wound, otherwise than by trickling water over it, for a whole fortnight.

At the expiration of this time, September 11th, chloroform being given a third time, the side-splint was changed for one of tin fitted to the back of the leg and foot. The foot was found slightly raised on the inner side, otherwise in good position; the wound was filled from the bottom and sides by coarse vascular granulations, not thoroughly united, so that three passages, admitting a probe loosely, ran to the posterior inner corner of the wound, where a small portion of the calcaneum was exposed (having apparently escaped the gouge); all other parts of the wound presented to the probe a softish mass, which it was not attempted to penetrate. The edges began to draw in and cicatrize, and their neighbourhood became much improved on the state prior to the operation.

About October 10th, Mr. Erichsen examined the wound, and found no bone exposed.

October 15th.—By Mr. Erichsen's advice, ointment of the nitric-oxide of mercury was used to the edges of the flabby wound with advantage. The anterior fistula healed, the posterior one became quiescent. A dextrine bandage was applied, and the patient discharged.

November 15th.—The wounds were fairly healed.

Christmas and Lady-day.—He is going on thoroughly well, can walk without pain; there is free mobility of the new joint, the cicatrix is becoming much firmer and smaller. Till now a splint has been constantly employed; he may have a boot fitted. Slight inversion of the foot continues, and the leg is about one inch shorter than the other.

June 14th.—Mr. Gray, of Cork street, made him a well fitted boot, the heel raised, an iron support up to the knee (jointed opposite the ankle to allow limited motion), with a broad strap around the ankle, and another band below the knee. The lad is able to walk and run without any pain, and with merely a halt, partly due to the incumbrance of the instrument. The foot is perfectly sound; he can extend it well, flexion of it on the leg is not so easy, the present relative position of the parts being more disadvantageous for this action than before; its mobility is complete. Inversion of the foot has disappeared.

I must acknowledge my thanks to Mr. James Turle, the house-surgeon, for the great care and ability with which he treated the patient.

A few remarks may be offered on this case; the full feasibility of the operation was fully established, supposing the disease to be confined to the astragalus, by the success of similar operations occasionally requisite in the after-treatment of dislocations of that bone. Had the calcaneum been more diseased than it proved to be, any portion demanding such treatment would have been removed by similar incisions, even if it had been necessary, following Mr. T. Wakley's example, to excise the whole of that bone. It is noticeable that no hæmorrhage occurred; no important tendons or ligaments were divided; and that the recovery is perfect.

That we may in future be able to *restore* the sound condition of scrofulous caries of bone is to be hoped for; at present we may congratulate ourselves if the excision is complete, and the functions of the part uninjured. No other apparatus than a high-heeled boot is now necessary.

In a case of Liston's, the astragalus and ends of the tibia and fibula were removed with success. Other cases of the removal of single bones of the tarsus for scrofulous caries, have been, I believe, limited to less important ones than the astragalus.

Chloroform is a most important agent, as the patient can be fairly examined, as he should be, some days before the operation.

The absolute necessity of not meddling with the parts after the operation, where interference is uncalled for, may be well illustrated from Stromeyer on Gun-shot Wounds: "A young officer, whose humerus had suffered comminuted fracture two-fingers' breadth below its head, and for whom exarticulation of the part had been first proposed, complained to me bitterly that his attendant had allowed the first dressings to remain so long, that maggots had bred in them. I told him, however, that he must thank this gentleman for the preservation of his arm."

In the dead body of adult males, I have since found that the astragalus can be removed by clipping off its head by Liston's forceps, and by dividing the body of the bone backwards by the same instrument, so as to cause no kind of injury to surrounding structures.

On referring to Mr. Dunn's remarkable case, I am no longer surprised that it is not especially quoted as an example to follow, as such repeated operations and dangers from hæmorrhage could hardly allow of success in the average of cases met with. The excision of the tarsal bones between the astragalus and metatarsal, viz., of the cuboid and ext. cuneiform, first, and of the scaphoid and other cuneiform, later, besides at the later period scraping the astragalus, and removing the tarsal ends of the second and third metatarsal—although the patient had four years afterwards a foot in which "the natural appearance was little altered,"—this wholesale excision is certainly a case of wonderful recovery, but at the same time is certainly—on account of the destruction of natural connections, and necessary division of tendons, vessels, &c.—a case which cannot be

quoted otherwise than as an exceptional one. I expect, therefore, though itself successful, it has actually been of more harm than advantage to conservative surgery, for few would like to undertake such an uncertain operation.

I am not aware of excision of the astragalus alone, for scrofulous disease, having been previously performed.

PATHOLOGICAL REMARKS

ON THE KIND OF

PALPEBRAL TUMOUR

USUALLY CALLED, IN ENGLAND, TARSAL TUMOUR.

BY

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Received Nov. 28th, 1853.—Read Jan. 24th, 1854.

THE subject of tumours of the ocular appendages is obscurely treated of by writers, more, I believe indeed, than any in the whole range of ophthalmological literature. The several stages of the same affection are described as different diseases, and the same diseases are dissimilarly delineated. A Greek and a Latin word bearing the same signification,—Chalazion and Grando, are applied to different morbid states. The disease I now propose to treat of is involved in similar perplexity; for I find it spoken of as "fibrinous tumour," "tarsal tumour not encysted," "albuminous tumour," and by other terms equally erroneous. There is also disagreement concerning its connection, whether moveable or not, and even as to consistence, whether hard or soft. I propose, therefore, in order to be understood respecting the tumour I mean, to describe the most palpable objective characteristics, before I point out what appears to be its pathological condition, and which has not, so far as I am aware, ever been demonstrated.

Commencing, then, with the external characters, I would speak of it as a hard, spherical, well-defined tumour, in size

varying from that of a grain of small shot to that of a pea, and limited to a position on the eyelid corresponding to the space bounded by the cilia bulbs, and the upper margin of the tarsus; that is, corresponding to the position of the meibomian glands, not growing at the edge, and immoveable. Inadherent to the skin, which may or may not be traversed by enlarged blood-vessels, being usually solitary, and for the most part growing on the upper eyelid, yet acquiring the largest dimensions on the under, where the skin is generally in the natural state; not unfrequently giving, on the internal surface of the eyelid, indication of its existence by a spot of preternatural redness, and at a later period discoloration, or even a small fungous growth.

In proceeding now to its pathology, I must at starting acknowledge the assistance that I have here received from Dr. Druitt. A very marked example of the tumour on the upper eyelid in a male, æt. 53, having come under my care in the summer of this year, I turned the skin aside and removed it, together with the corresponding portion of the tarsus, and sent it to him for investigation, as he was at the time working on the subject of tumours. It may be well, before I subjoin his valuable report, to state, that the wound was brought together with sutures, and healed quickly, not only without disfiguration, but without leaving a scar.

"The tumour," he says, "was oval, $\frac{17}{48}$ths of an inch in its length, and $\frac{13}{48}$ths in its short diameter; having on the one side the entire thickness of the tarsal cartilage (so called) with the conjunctiva, on which a few meibomian follicles projected in the form of yellow granules; and on the other, some fibres of the orbicularis. It was evident that the growth was most intimately adherent to the (so called) tarsal cartilage. On bisecting it by a clean incision, some viscid, puriform fluid escaped. On closer examination, the centre appeared to be constituted by a very clear transparent membranous cyst, almost $\frac{1}{18}$th of an inch in diameter, containing the aforesaid puriform fluid, and in the very midst, a small perfectly smooth circular pellet of sebaceous matter. Around this cyst was a soft pinkish material, and this again was con-

tained within a tough fibrous capsule, continuous with the fibrous envelope of the (so called) tarsal cartilage." He continues: "On making further sections, and examining them microscopically I perceived—1st, The conjunctival surface covered with epithelium, several branches of meibomian follicles projecting like villi, these follicles being filled with solid or liquid sebaceous matter, and constituting the yellow granular bodies visible to the naked eye. 2d, I noticed the proper fibrous tissue, commonly called cartilage, of the lid, the fibres for the most part running parallel with the conjunctival surface; it was abundantly permeated by vessels, and contained in spherical loculi, bunches of meibomian follicles. 3d, These follicles, except that some projected, as aforesaid, like bunches of currants, on the conjunctival surface were mostly contained in spherical fibrous loculi within the fibrous membrane. Some contained soft, others hard matter. 4th, The tumour itself, consisting externally of a dense, fibrous cyst, continuous with the fibrous tissue of the lid; within this a layer of fibro-plastic matter, soft, pink, abundantly supplied with vessels from the fibrous cyst, composed of fibro-plastic cells, with a very little intercellular fibrillary matter; within this, the thin pellucid cyst above mentioned, containing a puriform fluid, made up of pus globules, epithelium cells loaded with oil, and in the centre a perfectly round pellet of sebaceous matter." In conclusion, he suggests the following to be the order of development: "1st, The formation, with a meibomian follicle, of a pellet of hard sebaceous matter. 2d, The secretion of a more copious epithelium and fluid matter around. 3d, The addition of fibro-plastic matter around the obstructed gland follicle, distending the loculus of fibrous membrane into a cyst."

Through the liberality of the Museum Committee of the Royal College of Surgeons, I have been allowed, in conjunction with Mr. Quekett, to examine two tumours of this class belonging to the College Museum, whereby the accuracy of Dr. Druitt's statement is verified, and other facts have been elicited. It was quite impossible, while these specimens were in the bottles, to understand them, and the references

to them in the Catalogue, is not descriptive. I mention this, because I have, elsewhere, rather misrepresented them. In the one, on the outside of the tarsus from which the skin and the orbicularis muscle are removed, are two growths, one very small, too minute, perhaps, to have been recognised in life, and overlapped by the greater, many sizes larger; which, although firmly incorporated with the tarsus, holds its union by a small base. On the inside of the tarsus, the site of each is plainly marked by yellowish deposits in the course of the meibomian glands. Both were cut across, and found to contain epithelium scales and sebaceous matter. I beg to direct attention to the circumstance that the lesser, which is just enough developed to admit of a distinctive character, is equally well marked within the lid as the larger.

The other specimen afforded less definite information, yet it was peculiar and also instructive. The tumour occupied the entire upper eye-lid, from which the tarsus had nearly disappeared. It consisted, on the external or upper surface, of a dense fibrous sac: on the inside, that is within the lid, of conjunctiva. The interior, which was irregular and crypt-like, was, as in the other tumours, filled with epithelium scales and sebaceous matter. It would seem here, as if the entire meibomian apparatus had been simultaneously diseased.

I submit to the Society whether, if they consider the pathology of the disease to be proved, it would not be judicious to institute the term meibomian tumour, and to adopt a name alike simple, correct, and significant, an advantage not often to be met with in the nomenclature of ophthalmic literature, which is for the most part abominable and barbarous.

I have often heard it advanced in argument against the tumour originating in the meibomian glands, that it is always on the outside of the tarsus. This erroneous statement is advanced on the false supposition of the situation of these glands. It has long been pointed out that they are imbedded in the tarsus; however, anatomical works state differently,

and describe them as seated between the conjunctiva and it. The entire glands are within the tarsus, their ducts even traverse it, and open on its free margin. The relative anatomy of the parts may to a certain extent be seen with the naked eye, for if the tarsus be dissected out, the glands will be equally visible on either side.

It appears to me that the determination of the tumour outwards, depends on the same law that causes the elimination outwards of foreign substances from the body, and includes in its operation the directing of morbid growths to the surface. That there is occasionally an exception to the law, the tumour taking an inward direction, and appearing on the side of the eye-lid, all surgeons engaged in ophthalmic practice are aware. It then appears in an arrested state, which may be thus explained. The discoloured spot on the interior of the eye-lid, above spoken of as giving internal indication of existence of the tumour, is produced by absorption of the tarsus, solely, I believe, in consequence of the pressure produced by the tumour on the eye-ball, and hence the upper eye-lid, as it is more in contact with the eye-ball, usually exhibits this change earlier and in a more marked degree. This may not proceed beyond a very limited extent, but occasionally much of the tarsus is removed when the determination inward would seem inevitable. With the removal of the tarsus, the chief and densest covering is lost, and perhaps as a consequence, there is no deposit of the fibro-plastic material. The conjunctiva, then the only envelope, if not interfered with by art, is apt, like the tarsus, to suffer from absorption and ulceration, allowing the exposure of the distended follicle, which in turn is similarly affected, and discharges its morbid contents, or throws out a fungus growth.

It is well known that meibomian tumours may contain dissimilar substances, or a mixture of them; we meet with glairy, sebaceous, creamy, or purulent deposits; and that the amount does not always bear an uniform relation to the size of the tumour, there being sometimes scarcely any fluid in a very large one, the sac being filled with a solid material.

On this point I venture to suggest, that some of these characters depend on the changes effected in the fibro-plastic material that is deposited. For instance, with the onward development of the plastic material, white, or yellow fibrous tissue is produced, as in the tumour commonly called polypus; hence the more solid tumour. Or, if it degenerate and undergo retrograde metamorphosis, the cells are converted into pus, or pyoid cells, and the inter-cellular tissue into a creamy fluid.

P.S.—I desire to say, that since I have adopted the pathological views above stated, I have, whenever surgical measures are required, ceased to employ in general the usual method of attacking the meibomian tumour from the interior of the eye-lid; but for the most part, that is, when the tumour takes an outward direction, I divide it on the outside, squeeze out the contents, and when it can be accomplished, pull away the cyst with a pair of forceps, or if necessary, remove it by dissection. I am enabled to assert, that this process is far superior to the other, insomuch as it is instantaneously effectual. It is not necessary for me to point out the tediousness and uncertainty of the older method. I must add, that there need be no fear respecting the formation of a scar on the eye-lid; for if the incision be made horizontally, and the edges be brought together by a strip of plaster, no trace of the operation is left.

NOTICE OF A CASE

OF

SKIN DISEASE

ACCOMPANIED WITH

PARTIAL HYPERTROPHY OF THE MAMMARY GLAND.

BY

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Received Jan. 10th.—Read Feb. 24th, 1854.

THE record of an isolated case ought not to be laid before this Society without sufficient reasons in the way of apology. In diseases the pathology of which is in a great measure understood, and of which fresh knowledge can only be established by collating the facts of a variety of cases, the record of one, or even a few, remarkable incidents can serve but little to advance our clear appreciation of disease.

It is requisite, therefore, to the value of a single record, that the disease should be rare; that the appearances should have been unrecorded; and that there should be a satisfactory issue, whether that be of recovery, or of elucidation through the means of its fatal course affording a subsequent examination of the nature of the disease and of its relation to other classes better known.

I conceive that this case includes the earlier-named conditions requisite for the apology. The alarm which the appearances gave rise to were sufficiently grave to commend it to our careful examination; while the favorable issue, if less calculated than a fatal one to elucidate its history, at

least bears with it a certain amount of instruction on the point of treatment.

The subject of this case was a young lady, æt. 20, of fair complexion, light blue eyes, and fair hair. When I first saw her, the left breast presented a diseased surface, at the upper part, to the extent of about four inches in length by about an inch and three quarters in width. The appearances presented were a perfectly smooth, polished surface, of an opaque yellowish-white colour, like polished vellum or ivory; the margin of the diseased portion was defined by a strongly-marked border of injected vessels, but on the polished surface no vascularity could be perceived; there was no exudation whatever on any part of the breast—no crust or scurf of any kind.

The young lady had noticed the first appearance of this state of skin twelve months before, when it appeared about the size of a florin. It had been watched during the interval by Mr. Cartwright, of Oswestry, and had gradually increased during the last four months, assuming the appearance I have now described. The breast itself was larger than its fellow, and on examination by touch was found to contain several hard, resisting, nodulated tumours, varying from the size of a dwarf orange to that of a walnut, one of which (a small one on the left side) was alone sensible on being touched. A small enlarged gland was found in the left axilla, to which an absorbent vessel could be traced from the breast. The young lady suffered no pain in the part affected, but merely acknowledged to a sensation which she described as simply reminding her that there was something there. She was cheerful, and not suffering any serious apprehension; her general health was good, the constitutional change only being rather in defect.

Shortly after I first saw her, Mr. Ure joined me in consultation. The following description is quoted from his very accurate notice of the appearances, as recorded in his note-book:—"The integument over the upper part of the left mamma is thickened and indurated, or rather condensed, in an uniform manner, to the extent of four inches trans-

versely and one inch and three quarters from above downwards, and presents a dull white appearance, not unlike parchment; it is the seat, occasionally, of increased heat; one of the axillary glands is enlarged. . . . Some of the lobules of the subjacent glandular structures are enlarged, and of a more solid consistence than natural. There are several punctuate elevations of acne indurata over the back of the neck and shoulders." Mr. Ure was of opinion that there was partial hypertrophy of the mammary gland, with interference with the nutrition of the adjacent integument, connected with the catamenial disturbance.

It was determined to give the patient small doses of liquor potassæ twice daily, in infusion of cloves, and to have the affected surface pencilled over twice a week with tincture of iodine. She was recommended to return to the country for a month.

On her return to London, no progress having appeared towards recovery to a healthy state of the part, we acceded to the request of Mr. Cartwright, that Mr. Hodgson's experience should be added to the consultation. Mr. Hodgson compared the appearance to that of a scar left by a blister, as it appears after death. It is useful to record the impression which the appearance made on different observers. The vascular margin, which remained as at first seen, would, however, to my idea, have failed to establish this resemblance. The only case which Mr. Hodgson adduced as bearing any resemblance, was that of a woman, æt. 46, and, as that case ultimately proceeded to display itself as carcinoma, he was led to draw an unfavorable prognostic. With this discouraging view of the probable result, he deprecated the smallest approach to irritation of the part, and recommended a lotion of the diacetate of lead to be substituted for the iodine, to be applied four or five times in the day; the part to be covered with oiled silk. The general treatment was continued, and the patient once more returned into the country. At this time Mr. Ure's note is as follows: —"Parchment-like patch, as before, surrounded at its margin by a narrow faint blush, from capillary injection;

indurated and enlarged lobules of the mammary gland can be felt in the axillary half of the breast, and also towards the sternum, lying under the patch."

The diseased surface, however, continued to increase in extent, and with its spread the anxiety of friends became of course more serious. All parties naturally wished for the first surgical opinion in London, and I felt it right to comply with the suggestion of the friends, that I should have an interview with Sir Benjamin Brodie.

In his very large experience, Sir Benjamin Brodie could only adduce a single similar case—one which had occurred in the wards of St. George's Hospital, followed by a complete recovery. In that case the skin alone had been involved. The process of cure had been by throwing off of successive layers of diseased skin, during which the extent of surface became continually reduced, the skin beneath ultimately assuming its natural appearance, the patch becoming smaller and smaller, till it disappeared. Sir Benjamin Brodie considered the case less allied to carcinoma than to dry gangrene, since the vessels of the white surface were apparently destroyed, the injected edge forming a line of demarcation from which the vellum-like surface might be thrown off.

The favorable tendency of the experience afforded by the former case was corroborated by the fact that at this time the tumours in the breast were considerably reduced, although the gland in the axilla and its connecting absorbent vessel could still be felt.

The general treatment was but little varied. An alterative every other night, with the alkali in liquid extract of sarsaparilla; and glycerine was ordered to be rubbed on the part night and morning.

After an interval of six months we again saw the case. The surface of the breast had returned to its natural state, the patient describing that it had faded away gradually. Similar appearances had, however, shown themselves in various parts of the person—one on the inside of the left upper arm and others on the thigh: there was exactly the

same ivory-looking surface, and the same vascular margin. Thus all apprehensions subsided of anything more serious than a simple cutaneous disease, the alarming concomitant of the tumours in the breast having been plainly the result of the delicacy of the organ in which it first appeared.

The progress of the case afforded satisfactory evidence of the correctness of Mr. Ure's first opinion, formed as it was upon reasoning in the absence of experience. It also appears to me that, though there was no obvious throwing off from the edges of the scar, as the curative process anticipated by Sir Benjamin Brodie, yet that this very course of cure in all probability did take place may easily be supposed, and that the continued friction by the hand in applying the glycerine may have gradually removed small portions of the dead skin from the surface, as they became ready to be displaced by such mechanical means. It is only under some such supposition that we can understand how a skin so disorganised could have reassumed its natural vascular state.

Judging from the result, and from there never having been any apparently ulcerated surface, Sir Benjamin Brodie entertained doubts of the correspondence of the disease with that of the state of dry gangrene, to which he had to a certain extent compared it in its earlier stage.

This case is at least a rare one, since it has not come within the observation of men of large experience, and is not noticed in any works on skin diseases. The characters are somewhat analogous with those of squamous diseases, although one of the distinguishing marks of squamous diseases is absent, viz., the red spots with which the eruption commences. Another peculiarity of some of the squamous diseases, viz., the commencement of the healing process from the centre of the diseased surface, did not also occur. The chief point of similarity is the ivory-like state of the cuticle, in some degree analogous to a large scale.

To propose an explanation of the progress of the disease, I should suggest that the extreme vessels of the true skin appear at some commencing point to have undergone a state of engorgement. This state of engorgement is distinctly

noticed at the margin, which we may suppose to be an extension of the original point; from some cause, consequent on the engorgement, the healthy nutrition of the part appears to have been cut off, and the extreme vessels to have remained incapable of carrying blood. As the portion of destroyed vessels increased, the engorgement has continued to spread outwardly around it.

This outward spreading by an enlarged concentric margin is precisely similar to that of one of the squamous diseases (lepra). We cannot, however, trace the same process of a return to a healthy state as we do in lepra, in which last the renewal commences from the centre, where the disease originally began. In the case before us the scale remained entire, and disappeared by an almost imperceptible process. It is probable, had the case been left without local treatment, instead of the diseased cuticle being rubbed off by glycerine, it might have scaled off, as suggested by Sir Benjamin Brodie.

This point remains for future observation, on which account, as well as that it may be properly classed and recognised, it is desirable that any recurrence or variety may be communicated by the members of this Society.

CASE

OF

MOLLITIES OSSIUM,

PRECEDED BY

DEGENERATION OF THE MUSCLES.

BY

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Received Jan. 26th.—Read Feb. 14th, 1854.

Mary G— was admitted into St. Mary's Hospital, under my care, March 26th, 1852. She was twenty-six years of age, unmarried, and had never been able to follow any avocation, on account of weak health. She was about four feet ten inches high, and six stone seven pounds in weight, not emaciated, and of symmetrical figure. A waxy, yellow complexion, with bright scarlet colour in her cheeks, gave her the aspect of a delicate person. No history of hereditary disease of any kind could be elicited from herself, or from enquiries made at her native village. The ankles were œdematous, and she walked slowly, as if from languor, on a flat surface; when she attempted to ascend steps or to raise herself from a stooping posture, defective power in the muscles of the haunch and thigh became very evident. The flesh of the whole body was exceedingly soft and flabby, the calf hanging down in the baggy way that it does in emaciated persons. She stated that she had first become an invalid seven years previously, her illness commencing by weakness and pain across the loins, especially at the end of the short ribs on the left side. The same symptoms had continued, with occasional variations, up to the date of admission. She

usually felt worst in early spring, and got better during summer and autumn. The bowels were somewhat costive; the catamenia generally regular, but occasionally postponed for a fortnight beyond the customary time. The urine, examined on admission and frequently while she was in the hospital, was variable in quantity, and variable in specific gravity, in proportion to its quantity, from 1·020 to 1·028. It was sometimes neutral, rapidly turning alkaline; but generally acid, depositing a considerable sediment of lithate of ammonia, soluble in heat. In all cases, boiling immediately threw down a cloud of phosphates, soluble in mineral acids, and showed the absence of albumen.

With the exception of a stitch under the short ribs of the left side, caused by turning in bed, and a sense of great debility in the back on standing, there was no pain experienced at any time, either with or without pressure.

The patient remained in the hospital, taking steel, for five weeks, without any change in the symptoms except the disappearance of œdema and improvement of appetite; after which she left at her own desire. She returned in three weeks, and again remained under observation for ten days, when she was advised to go to the seaside, and left with the expressed intention of doing so. During her residences at St. Mary's, the bones of the back and limbs had been carefully examined several times, without any deviation from the natural state being discovered, except that the ribs on the right side, viewed from behind, were not quite symmetrical, being more prominent than the left. On her going away, I told her of the difficulty experienced in arriving at a diagnosis, and desired her to let us know when any medical man could discover the nature of her complaint.

From St. Mary's she went to St. George's, and was under the care of Mr. Cutler for about six weeks; after which she left at her own desire. The symptoms continued equally obscure at St. George's, and no diagnosis of the case was formed till one night spontaneous fracture of the left femur occurred. She was then removed to her home, at Islip, Oxfordshire, and placed under the care of Mr. Blick. She

informed him of the wish I had expressed to learn the result of the case, and through his kindness I am enabled to detail the remainder.

It appears that when he saw her on the 11th of August, 1852, a fortnight after leaving St. George's, fracture of both femora had taken place in the upper third of the bones. Obtuse angles were formed at the seat of fracture, by the thighs being drawn upwards and outwards, and twisted on themselves, so that the external border of each foot lay on the bed, and the soles approximated to one another. The only parts of the lower extremities capable of voluntary motion were the toes. The shin-bones felt soft on pressure, producing a sensation to the finger described by Mr. Blick as like that of a fibro-cartilaginous tumour. She breathed, ate, drank, slept, excreted fæces, urine, catamenia, as well as usual; taking meat, wine, and beer, but refusing medicine. She had no pain, except when the tumefied parts about the fractured ends of the bones were touched, and those were excessively sensitive. No important change seems to have occurred—except that the body kept shortening, and the leg-bones getting softer, so that the foot could be raised three or four inches from the bed without altering the position of the knee—till April, 1853, when the right arm became painful to the touch and paralytic.

In May, the same misfortune happened to the left upper extremity also.

In June, the pelvic arch gave way, the mons veneris being drawn upwards and the anus thrust forwards, the alæ of the ossa ilii falling inwards.

In July, the ribs on the right side gave way, and she began to suffer much from dyspnœa and cough, with quick pulse, fever, and restlessness.

In August, the bones of both arms were found quite soft.

In September, the ribs on the left side fell in, and she was now much distressed by increased dyspnœa and palpitation of the heart. The contractions and dilatations of that organ were distinctly visible through the fleshy parietes. The lower jaw and bones of the skull also felt soft on pressure.

Towards the end of October the distortion of the lower parts of the trunk was so great, that the fæces could not be naturally expelled, and had to be removed by mechanical means.

She at last died of dyspnœa, on the 6th of November.

Several times during the illness, Mr. Blick sent me some of the urine secreted by Mary G—. It presented always very similar physical characters to that passed in St. Mary's. Whilst acid, the microscope showed a great quantity of lithate of ammonia, vesical epithelium, and a few crystals of oxalate of lime. After it became alkaline, there were to be seen in it a number of yellow spherules, some of them furnished with thorn-like processes, which Dr. Hassall informs me he considers to be uric acid in combination with an earthy base. There was also a large quantity of vesical epithelium, numerous crystals of triple phosphate, and a few stellæ, pronounced by Dr. Hassall to be phosphate of lime. A quantitative analysis of this urine, by Dr. Beale, gave the following result:

	Urine of Mary G—.	*Healthy Urine (Berzelius).*
Water	971·00	933·00
Solid Matter	28·10	67·00
	In 100 *Parts of Solid Matter.*	
Urea	17·7	44·7
Extractives	36·3	39·0
Lithic Acid	0·6	1·4
Earthy Phosphates	3·3	1·4
Fixed Alkaline Salts	41·9	25·9

It will be seen, therefore, that the solid matters of the urine were diminished below the natural standard by more than half, and that this diminution was due to deficiency of animal matter; that the alkaline and earthy salts were nearly doubled in amount, the principal comparative augmentation taking place in the earthy phosphates.

After death, the body was found to have shrunk from four

feet ten to three feet one inch and a half, and could be rolled up upon itself, to use Mr. Blick's forcible expression, "like an ill-stuffed bolster." No examination of the viscera was made, but portions of the tibia, sartorius, and rectus femoris muscle were removed, and a sharp instrument passed into many parts of the bony skeleton, which was found universally soft and unresisting.

The section of tibia sent to me was of the colour of muscle, soft and friable throughout, presenting to the knife scarcely more resistance than brain, and retaining its shape solely by the aid of the tough periosteum. No remains of bone could be felt except on cutting the periosteum, where a slight grittiness was perceived on making a section with sharp scissors for microscopical examination. Under the microscope, the whole of the bone, to within half a line of its external surface, was seen to consist of large fat-vesicles, containing, some white, others a reddish oil, and thus accounting for the colour of the texture, with the intervals between them filled up with spherules of various magnitude, mostly about as large as blood globules, of a dull red tinge. They were formed apparently of aggregated grains, and some had an indistinct nucleus. No fibrous structure could be detected in this situation.

The part next to the periosteum, which felt gritty when under the scissors, exhibited, when examined under a quarter-inch lens, small islands of opaque bone, which, however, did not retain a perfectly healthy appearance; the bone corpuscles being indistinct, and the caniculi not to be discerned. These islands were surrounded by some structure more transparent, and that again bounded by a reddish fibrous structure, in which were oil globules of various sizes, and a few oval fat-vesicles towards the inside. The addition of hydrochloric acid caused a slight disengagement of gas.

The portion of rectus muscle was, to the naked eye, of the natural colour, but of too homogeneous an appearance. Under the microscope, it presented no traces at all of fibrous structure, or even linear arrangement. It had become a mere con-

geries of fat-vesicles, the interspaces between which were filled up with globular granular corpuscles of various sizes, estimated by Dr. Seiveking as from $\frac{1 \text{ to } 2}{5000}$ths of an inch in diameter. Many of the larger had a granular nucleus.

The circumstances which have induced me to lay this case before the Society are the following:

1st. The portrait which is afforded of an early stage of the disease—a stage at which it is rarely the subject of observation.

2d. The impression produced by it upon my mind that the degeneration of the bones was preceded by that of the muscles—that the degeneration of the two tissues was dependent, in this instance, on the same crasis, and the probability, therefore, that such is its history in other cases also.

3d. The opportunity of placing on record a careful quantitative analysis of the urine in this disease.

4th. The fact of the degeneration being least advanced in the external circumference of the bone.

5th. The formation of perfect fat vesicles in both bone and muscle.

As respects the symptoms which precede the known softening of the bones, attention may be called to the absence of those rheumatic pains which are usually stated to be precursors, nay by some supposed to be the actual causes of the malady. When the disease is fairly established, these pains may conjecturally be referred to the pull which healthy muscle exerts on the periosteum, deprived of its usual firm base of resistance. But, as in the case before us, the muscle appears to have yielded first to the morbid influence, it did not strongly contract, did not drag on the periosteum, and so no pains of consequence were experienced. We see, also, that previous to the softening being demonstrated to exist, the same appearances were noted in the urine which were observed when the disease was fully established, pointing to a great probability that the chemical constitution was not dissimilar. We see also that the degeneration was purely automatic, not arising as a consequence of any other morbid state, nor, as far as could be ascertained, of an hereditary taint.

That the degeneration of the muscles preceded that of the bones is of course a matter of opinion. A person observing the circumstances only after death, would naturally suggest that it was due to their necessary inertness for so many months. But the condition of the patient during her stay at St. Mary's discountenances this idea. The mechanism of the bones was complete so far as their ordinary uses demand, yet so peculiar was the partial paralysis, or rather torpidity of fibrous contraction, that, in spite of the rarity of the disease, I was induced to select fatty degeneration of the muscles as the only explanation I could give.

As respects the quantitative analysis of the urine, it is confirmatory of the suspicion usually expressed, though not hitherto proved, that a great loss of lime takes place through this channel.

The observation that the degeneration was least advanced in the external surface of the bone, shows that its course is from within outwards; and that, therefore, till the shell of osseous structure bends or breaks, the bone is as useful as ever for the purposes of muscular motion. The mere thinness of the plate of bone remaining intact can make no difference mechanically to the action of the muscles implanted in it, so long as it is strong enough to bear the strain. It will be seen that this last argument has an important bearing on the first observations made concerning the early diagnosis of the disease.

ON THE

KELOID OF ALIBERT,

AND ON

TRUE KELOID.

BY

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Received Feb. 16th.—Read Feb. 28th, 1854.

THE term *keloid*, or *kelöide*, the name given to the singular affections of the integument about to be described, has been variously interpreted; some deriving it from κηλη, a tumour; others, in reference to certain supposed resemblances, from χηλη, a crab's claw; or from χελυς, a tortoise; whilst others, apparently with much greater propriety, derive it from κηλις, 'quasi ustione facta macula,' the disease in every instance presenting a greater or less resemblance to some one of the diversified effects left by a burn.

The more immediate object of this very slender communication, is to show that the keloid originally described by Alibert, and now so generally recognised, is altogether different in its mode of development, character, and progress, from another disease occurring in the same tissue, and to which, with much greater aptitude, the term keloid may be applied, if we are to regard resemblance to the effects of a burn as its correct interpretation; for I think it will be shown, that whilst the keloid of Alibert and others can hardly be regarded otherwise than as a fibrous tumour developed in the subcutaneous areolar tissue, the other form of disease to which I have alluded, although originating in the same tissue, is of a character and leads to consequences

widely different. In order, however, to illustrate and confirm this proposition, it will be necessary to give a description of both diseases; and in so doing, I will, as far as possible, avoid trespassing too much upon the time and attention of the Society.

I propose distinguishing the two diseases in question by the terms "*Keloid of Alibert*," and "*True Keloid*."

KELOID OF ALIBERT.

I have given the name "Keloid of Alibert" to this form of disease, because I believe Alibert to have been the first to discriminate and accurately describe it. In his celebrated work, 'Description des Maladies de la Peau,' will be found a very accurate representation of it, executed with all the artistic skill, and perhaps a little of the exaggeration of colouring, for which that work is so remarkable. He there suggests its holding a middle place between what he so vaguely and indiscriminately calls "dartre" and cancer, and was led in consequence to give it the name of "cancröide," like cancer; further justifying the appellation, however, by comparing, as others have done, the claw-like rays or processes of the extending disease to the claws of a crab. Since the period of Alibert's original publication, several other writers have furnished cases and commentaries to illustrate the character, progress, or pathology of the disease. Amongst these we find the names of Biett, Velpeau, Cazenave, Coley, and others; but by far the most complete and elaborate essay on the subject has only lately been written by Dr. Dieburg, of Dorpt, and published in the 'Deutsche Klinik' at Berlin, and for a knowledge of which I am indebted to my colleague Mr. Birkett and Dr. Whitley.

The keloid of Alibert first appears in the form of very small, hard, shining, tubercular-looking elevations, of a roundish or oval shape, somewhat firmly set, of a dusky or deep red colour, and generally attended with itching or pricking, shooting or dragging pains in the part. These tumours slowly increase until they attain a height of two or three

lines, and comprise an area varying from that of a horse-bean to that of a small almond. So long as they continue to be abruptly prominent, the summit, or even the entire surface of each tumour, instead of remaining uniformly red, not unfrequently presents a pale or blanched appearance, as if from pressure of the increasing tumour upon the cutis situated above it, and which might at first sight be mistaken for some sort of fluid effusion. On close inspection, however, it is found, that so far from this being the case, the tumour displays a hardness, firmness, and elasticity, which almost convey the notion of so much fibro-cartilage, to which indeed it has been not unaptly compared. After an uncertain period, these hard shining tumours become broader, of more irregular outline, and occasionally slightly depressed in the centre. At this time, and sometimes even earlier, by the aid of an ordinary magnifying glass, or by the naked eye, delicate whitish tendinous-looking lines may be perceived, stretching across the surface of the tumours, mingled with minute blood-vessels of a bluish, purplish, or pinkish colour. The extension of each individual tumour now seems to be effected by certain tapering claw-like processes of seldom more than from half a line to a line in breadth, and probably from a quarter of an inch to as much as an inch in length, proceeding from the edges or angles of the expanding tumour. These claw-like processes appear to produce a puckering of the skin; and, as it were, draw the healthy integument into which they pass, towards the original excrescence, and within the influence of the local changes; appearances, nevertheless, which are probably the mere consequences of the stretching and dragging of the integument occasioned by the increasing size of the tumour beneath.

The slow and gradual increase of these tumours may proceed for months or years, and at last attain a size of an inch, an inch and half, or two inches in length, as much as half an inch or an inch in breadth, and probably an elevation of three or four lines above the level of the surrounding skin. There may be but a single tumour, or there may be several: when more than one, they may be congre-

gated together in the same neighbourhood, or may occupy parts of the integument remote from each other: when of the largest size, the tumour may so stretch and attenuate the integument as actually to protrude beyond it, exposing a red shining excoriated looking surface. The development of the tumour is occasionally preceded or accompanied by heat, and some degree of puffiness or tumefaction of the surrounding parts, but without redness or other discoloration; a state of things, indeed, which may temporarily supervene at any period of the disorder, either in consequence of some accidental cause of general excitement, some irritation applied to the tumours themselves, or spontaneously, and without any very appreciable cause whatever.

From the very commencement, as has been already observed, the disease is attended with itching and pricking sensations, which, as the former increases, are aggravated to a sense of constriction, or to severe pricking or stabbing pains, which prove extremely distressing to the patient. Under such circumstances, pressing or handling the tumour is loudly complained of; the sufferings of the patient, if a female, are not unfrequently such as to harass her during the whole of the day, and almost completely to deprive her of rest at night.

The morbid deposit which essentially constitutes the keloid of Alibert, takes place in the subcutaneous areolar tissue, between the cutis and adipose membrane. The occasional heat and tumefaction of the neighbouring integument, as well as the itching pain and redness of the tumour itself, sufficiently attest that the morbid process is at least accompanied by a degree of vascular excitement nearly allied to inflammation, an inflammatory state which, it would appear, gives rise to a certain amount of adhesion amongst the meshes of areolar tissue around; and, as we know that tumours of considerable size may be developed in the subcutaneous areolar tissue without either uneasiness, pain, or any very obvious change in the appearance of the skin itself, I am inclined to attribute to this accompanying inflammatory and adhesive process, the fixed condition of the tumour, the

great vascular injection of the superincumbent skin, and the intensity of the local pains, as well as those remarkable puckerings of the integument which attend the increase of the tumour, and constitute the claw-like processes from which some have derived the name "keloid."

The disease most frequently attacks females from the age of 18 to 35 or more, and in a large majority of instances is found situated near the sternum, between or upon the mammæ; it nevertheless occasionally affects the male, and in both sexes has been known to occur on other parts of the body, as the arms, shoulders, neck, belly, or even the head or face. Alibert, as already observed, considered it in some way allied to cancer; an opinion unsupported by any facts with which I am acquainted; whilst others, with perhaps no better evidence, have attributed the predisposition to a scrofulous taint. The development of the disease in different parts of the integument at the same time, or in succession, and its almost certain recurrence after extirpation by the knife or by caustics, clearly point to some peculiar constitutional condition; but what that condition is remains to be ascertained. All that we at present know respecting the exciting cause of the disease, amounting to no more than the fact, that, instead of arising spontaneously, on parts to all appearance previously sound, as is commonly the case, it has not unfrequently been observed to be developed upon and apparently excited by a cicatrix, as of a burn, a boil, or a recent wound, such as that inflicted by the punishment of flogging. To the disease, when occurring under the latter circumstances, Alibert, in a subsequent work, applied the term *spurious* or *false keloid*—the *cicatrix keloid* of Dieburg—a form of the complaint, however, which is sometimes altogether painless.

Case I. (Pl. 158^{50}, Model 231^{10}, 231^{11})[1] reported by Mr. Pratt.—Susannah Black, æt. 18, a single person, who has been residing with her mother, at Snowsfields, was admitted on the 6th October, 1853, having been transferred from

[1] The references are to plates and models in Guy's Hospital Museum.

No. 5, Mary, by permission of Dr. Babington, under whose care she had been since the 14th ult.

She is below the middle height; has dark hair, eyes, and complexion; a narrow forehead and heavy expression; but seems intelligent and is highly hysterical, and was formerly apprenticed to a laundress, but not strong enough to continue this occupation.

Her catamenia first appeared at the age of 15, and have recurred regularly since, generally continuing about three days, but with pain in the back and loins, and during the last two years with clots, sometimes of the size of a shilling.

Her father died of diseased heart, but the other members of her family are healthy, and none of her relations have ever suffered as she now does.

She is marked by the smallpox, which she had when three or four years old, but does not look unhealthy, and states that she was always in good health until about two years ago, when, from exposure to cold at Gravesend, while lightly clad, she first became ill, with pain in her head and right side, and at the scrobiculus cordis, shooting thence to the back. Six weeks after this, in Berkshire, having been gradually getting worse in the meanwhile, with loss of appetite and increase of pain, which for a time was so severe as to keep her in a bent position, but occasionally left the scrobiculus cordis and appeared in the loins, she suddenly vomited about a pint of dark clotted blood, after which she became better, but did not lose the pain in her back, and suffered from palpitation of the heart. About three months after this, having returned to London in the interim, the vomiting of blood recurred, and from this time was repeated at intervals, sometimes of two or three months, at others of two or three weeks only, until a few days before admission; and once in the hospital, about two weeks since, she brought up a teacupful of blood.

About twelve weeks since she had a gathering in her right breast, which discharged a small quantity of matter; two weeks after, and just as this was healing, her neck, chest, and both breasts swelled a good deal, with a dull

aching pain, but without œdema; one week after this, or two or three days after the swelling had subsided, she first noticed two small red pimples on the right breast, at its upper and inner part, which were painful, with a pricking sensation, and tender. Then, about one week after, two other similar raised spots, appeared on the left breast, at about the same position, but not symmetrical, and then two smaller ones above these; these all gradually increased in size; but in varying degrees, and, as they did so, at certain stages of their existence became white (?)

There are at present two raised spots on the right breast, nearly oval in shape, and of considerable size; four on the left breast, two large and two small; one on the upper part of the sternum; several at the upper part of the abdomen; and one on the left shoulder; and a cluster of equivocal white spots at the lower part of the back on the right side.

They seem to be in every stage of existence; some small, red or white; others of varying size, more vascular, generally of a red colour, and marked with small venæ, and traversed by peculiar white lines; but they all change colour occasionally (?) from white to red or even purple, and have a peculiar, firm, and unyielding feel. They have always a dull and aching sensation, converted into a more acute pricking pain by pressure; are more or less raised above the level of the surface, the largest as much as one eighth of an inch, or even more; have irregular margins, much resembling the contraction of a cicatrix, and appear to increase in size by an extension of the white lines which traverse them into the surrounding tissue, like feelers, to which, indeed, their irregular margins are due.

Her chest is well formed, her nutrition good; she seems to be subject to boils; has old cicatrices of venesection on each arm, and a small hard nodule on the left side of the neck, just above the sterno-clavicular joint, resembling an enlarged gland. Her tongue is white and moist; her pulse 80, full and regular; her countenance rather flushed; her bowels, which have been much relaxed, now act about three times daily, the motions being very loose; her appetite is bad;

she complains of pain in her head, across the top. The sounds of respiration and of the heart are normal, as well as the resonance of the chest on percussion, but the heart's impulse is strong and heaving, and the pulsations of the aorta felt above the sternum.

Case II (Model 229, pl. 158[57], pl. 158[54]), furnished by Mr. Whateley, surgeon, of Berkhampstead.—William Garrett, æt. 37, applied to me, about May, 1851, with a small tumour on the skin of the left breast, slightly elevated above the surrounding skin, silvery red in appearance, exquisitely tender, and about one inch in diameter. I recommended its removal, to which he would not then consent. On seeing him about a month afterwards, there was a second appearing, about an inch from the first, and subsequently a third. Such being the case, and fearing that others might still appear, I did not think it advisable to press the operation. He was then sent to Guy's Hospital, at the request of the late Bransby B. Cooper, Esq., in order that a model, &c., might be taken of the tumour in its then state.

After remaining some time, he again came into the country, and was under my care at the West Herts. Infirmary.

The tumour still continuing to grow, and the three having coalesced into one, and having no appearance of any fresh growth in the neighbourhood, I again advised an operation, to which he consented, and I removed it on the 10th of May, 1852, removing with it about a quarter of an inch of the sound skin all round, and fully down to the bone. The wound was dressed with warm water dressing and oil-silk, and was cicatrized. The cicatrix is now sound, and the man in good health.

The tumour, when freshly cut through, in structure, colour, and appearance most nearly resembled a cow's udder.

The slight sketch, No. 4, represents the result of a microscopic examination of the tumour, made, however, under very unfavorable circumstances, by Dr. Habershon, of Guy's Hospital.

A more minute and careful examination of a keloid

tumour has been supplied by Dr. Dieburg, of whose account of it the following is a translation:

"On section we observe a dull white colour, a dense tissue in which fibrous structure is visible to the naked eye, and a creaking sound is produced by the knife. On pressure, no fluid exudes in most cases; in a few, a watery fluid is seen, sometimes reddened by blood. This is characteristic, as different from the 'tumores verrucosi cicatricum' of C. Hawkins, from which a peculiar fluid may generally be expressed. Microscopical examination shows the different stages of development of the cells and fibres. We distinguish—1. More or less rounded bodies, the largest 0·05 of a millimetre; in their interior, we see a nucleus, and frequently other molecules. 2. Cells elongated in the direction of one of their diameters, in great numbers: they seem to constitute a characteristic element of all the tumours of 'cicatrix-keloid' (spurious keloid of Alibert). These cells, called by Follin 'elliptical bodies,' are rounded at their extremities, and their sides present central bulging. These cells are about 0·01 millimetre in breadth, and 0·06 in length. They contain a nucleus easily distinguishable by its brightness from the dull surrounding parts. 3. Spindle-shaped bodies, bulging in their centre, and having long, waving appendages. 4. Fibres of cellular tissue and elastic fibres. The fibres of cellular tissue are formed into bundles, which cross each other, and constitute a pretty dense web. The elastic fibres are less numerous and larger than the latter, and are not easily seen without immersion in acetic acid. When a slice of keloid in an early stage of development is placed under the microscope, it is found to consist almost entirely of the spindle-shaped bodies; at a somewhat later period these are seen to have lost their nuclei, and assumed a fibrous appearance: this is most frequent. At a still later period, we see distinct fibrous bundles, crossing each other, and by immersion in acetic acid, the elastic fibres become visible. The whole is nourished by a comparatively small number of blood-vessels. The surface is covered by a very thin layer of epidermis, consisting of tesselated cells, very

closely pressed together, which require softening before they become visible under the microscope."

The following translation from M. Labert's 'Traité practique des Maladies Cancereuses, et des affections curables, confondues avec le Cancer,' will probably not be considered out of place.

"Among the cases of spontaneous and multiplied keloid that we have observed, there were two especially curious, in consequence of their multiplicity and extent. In one case, under M. Velpeau, at "La Charité," the whole pectoral region of one side was covered with these tumours; many of which were sufficiently large to have reddened and eroded the surface of the skin at their borders.

"In the second case, a child æt. $10\frac{1}{2}$, had a very great number of keloid tumours, developed upon its back, red on their surfaces, and which had formed in the cicatrices which were consecutive to numerous applications of caustic potash, applied to the poor child by a charlatan, who promised to cure, by this method, a scrofulous disease under which the child laboured."

I may add to this passage from Lebert, the fact, that I have myself very recently been consulted in the case of a young lady of about eighteen years of age, upon whose back, shoulders, and breast, I counted as many as thirty keloid tumours. I was told that they originated in the cicatrices of boils which broke out about six or seven months before. From the situation, it had been a case probably of *acne*.

In regard to treatment little can be said. Various internal and external remedies have been tried in vain; and when extirpated by the knife or destroyed by caustics, the disease has, I believe, very generally returned on the seat of the original disease. When, however, the disease has been first developed in a cicatrix—the spurious keloid of Alibert—extirpation has proved more successful, the disease not having again made its appearance in several instances. It has indeed been asserted that the keloid tumour may subside spontaneously, leaving behind a white and depressed cicatrix; but I

believe this to be extremely rare, and is in itself a very improbable event, after the tumour has attained any considerable size.

TRUE KELOID.

What I have ventured to call "True Keloid" presents a very remarkable character, and leads to much more serious consequences than the keloid of Alibert. It is a disease, too, which, so far as I know, has not hitherto, with the exception of a slight allusion of Dr. Coley, been either noticed or described by any writer. Like the keloid of Alibert, it has its original seat in the subcutaneous areolar tissue, and is first indicated by a white patch or opacity of the integument, of a roundish or oval shape, and varying in size from that of a silver penny to that of a crown piece, very slightly or not at all elevated above the level of the surrounding skin, and probably unattended, in the beginning, with pain or any other local uneasiness or inconvenience, although a more or less vivid zone of redness surrounding the whole patch, or a certain amount of venous congestion in its immediate vicinity, sufficiently attests the vascular activity or inflammatory process going on in the parts beneath. Occasionally, and especially when the original white patch is of considerable diameter, its surface presents here and there a faint yellowish or brownish tint communicating to the whole spot a somewhat mottled appearance. The slow and insidious change taking place in the areolar tissue either stops and the spot disappears, or it proceeds, and at length begins to declare itself by a feeling of itching, pain, tightness, or constriction in the affected part, and frequently by a certain amount of subcutaneous hardness and rigidity, extending beyond the site of the original superficial patch, although as yet without any necessary change in the appearance of the superincumbent skin. This hardness and rigidity can be distinctly felt, and, especially when situated on the extremities, may sometimes be traced along the course of the neighbouring tendons or fasciæ, or stretching like a

cord along the limb, so as to bend or shorten it, and even interfere with natural progression. At length the part originally affected becomes more or less hide-bound, and a similar change taking place around the more superficial fasciæ and tendons, the latter become so tightened, fixed, and rigid, as to be no longer capable of performing their proper functions, and to such an extent, that the whole of a limb, but especially the fingers, may be permanently contracted, bent, and rendered almost as hard and immoveable as a piece of wood; thereby impeding progression, distorting the gait, and making the patient, a poor miserable cripple for the remainder of his life.

As these changes proceed, the patient continues to experience itching, pain, or a sense of tightness or constriction of the parts, till at length the disease begins to tell upon both cutis and cuticle. The skin, which may have previously presented only a slightly drawn or puckered look, imparting, to a greater or less extent of it, a ray-like appearance, now shrinks or shrivels; it assumes a dry, smooth, or glistening aspect; it undergoes a more decided change of colour, becoming reddish, pinkish, yellowish, or of a dead leaf colour; the cuticle exfoliates; the cutis manifests a tendency to superficial ulceration or excoriation, with consequent scaliness or scabbing, or, when not excoriated, is occasionally surmounted by obscure tubercular or nodular elevations—the whole appearance very closely resembling the remains of an extensive and imperfectly cicatrised burn. From some part of the boundary of the discolored and shrivelled skin, there may now and then be seen reddish, elevated, claw-like processes, of from half an inch to two inches in length, extending into the sounder integument, and bearing a very exact resemblance to those mentioned as being so characteristic of the keloid of Alibert. It must also be observed that, during the progress of the disease, it is by no means uncommon to find, scattered over various parts of the apparently sound surface, certain oval or roundish and flattened tubercular-looking elevations, which are somewhat hard to the touch, about the size of a split

pea or horse-bean, and without any other discoloration than what appears to be the result of accidental friction or irritation.

The above description of true keloid clearly points to some morbid change slowly taking place in the subcutaneous areolar tissue, whilst the itching, pain, and uneasiness experienced by the patient, the red zone surrounding the patch, and the injection of the neighbouring veins, as well as the subsequent appearances presented by the parts affected, would indicate that the morbid process going on in that tissue is one very nearly allied to inflammation, probably of a strumous kind. It would also appear that the inflammatory product, by its subsequent contraction, seriously interferes with the proper nutrition of the cutis, fixes it more or less firmly to the parts beneath, and, when deposited in the immediate neighbourhood of fasciæ and tendons, may, probably, after the lapse of months or years, lead to all those serious inconveniences which I have already described.

I will not abuse the patience of the Society by entering into any speculations respecting the origin and essential nature of this very singular disease; neither is it necessary to dwell upon plans of treatment, further than to observe that, with the exception of iodine, none of the many remedies tried, seemed, in extreme cases, to make the slightest impression upon either the appearance or the progress of the disorder. In one instance, however, less advanced, iodine, taken internally, with the simultaneous application of iodine ointment to the affected parts, did appear to arrest the advance of the local changes, and somewhat lessen the rigidity of the affected tendons. Whether the preparations of iodine administered at a very early period of the disorder would prove more effectual, I have had no opportunity of ascertaining, although I am inclined to entertain a strong opinion in its favour.

The following case presents an example of the disease in its earlier stages:

Case III (Models 222, 223, 224, Pl. 158[52]), reported by Mr. Towne.—Eliza Watkins, a young woman between 19 and 20 years of age, of ruddy complexion, fleshy and well looking, with light eyes, and hair tending to red, presented herself amongst the out-patients of Guy's Hospital early in June last.

She was in the situation of lady's-maid, and had for some time been residing at Cheltenham. Her general health was good, and at this time apparently undisturbed. She had been suffering from pain and stiffness in the left arm and left leg, for which she was now seeking relief.

The first appearance of the disease had been noticed twelve months previously, when a small white spot, about the size of a shilling, was observed on the left side; but, as neither pain nor inconvenience accrued, no anxiety was felt with reference to it until about eleven weeks prior to her appearance at the hospital, when she first became sensible of pain, attended with a dragging sensation in the left arm and left leg, both limbs being affected simultaneously. Medical assistance was now called in; poppy fomentations were ordered, and for some time persisted in; the disease still making slow but steady progress.

The lady with whom she was living, having occasion to visit London, brought the young woman with her, and took the opportunity of having a second opinion. The case was now treated as a sprain; but the patient, not feeling satisfied, determined to come to the hospital.

The two limbs were in a very similar condition. At this time they presented to the eye but slight indications of the disease, which principally consisted in a hard, drawn, tight look, on the limb being extended; there might, however, be felt, through nearly the whole length of both arm and leg, a rigid band, which gave to the touch the impression of some inelastic substance tightly strained under the integument.

The shoulders presented a mottled appearance, and had several whitish patches interspersed with numerous small tubercular-looking growths. There also existed a chain of

spots which nearly surrounded the right nipple, and several others about the neck and breasts. The spot on the left side (described as the first appearance of the disease) had now attained the size of a five-shilling piece, and had thrown out a band upwards towards the cartilage of the ribs, and a second descending towards the pubes.

During the second week in August, I again saw the patient. The pain in the arm and leg had much increased, with "a feeling of shortening" in the limbs affected; and, after sitting for some time, it was with difficulty the foot could now be extended. The band down the arm had become more distinctly expressed, had assumed a slightly tendinous and glistening character, and had thrown out several small lateral processes. A fresh spot had appeared on the upper lid of the left eye, and a second on the outer side of the right leg. Those on the shoulders had become more evident; the larger one had increased in size, become yellowish in colour, glazed on its surface, was hard to the touch, and did not move freely with the surrounding integument.

The next case exemplifies a more advanced stage of the disease:

Case iv (Model 225, Pl. 158[46]), reported by Mr. King. —Louisa Burston, æt. 11, was admitted, under Dr. Addison, December 8th, 1852.

The patient, who is a very strumous-looking subject, was very strong and healthy as a baby, but was noticed to be slightly ricketty when she began to walk; this was between eighteen months and two years of age; but when she was three and a half or four years old she had nothing remarkable about her.

From this time her mother always considered her delicate; but, beyond frequent attacks of ophthalmia, which have deprived her of most of her eye-lashes, and appear to have been of a strumous character, she has never suffered any decided illness.

Attention was first directed to the right thigh, about

fourteen months ago, on account of complaints on the part of the child of itching in that situation; and this appears to have been so intense, that measures were taken, by tying her hands, &c., to prevent her flaying herself. When first examined, red spots, like flea-bites, were observed thickly studding the inner part of the thigh, about its middle third, but not imparting any feeling of elevation to the finger.

This condition lasted about a fortnight, and was then succeeded by a flaky desquamation of the cuticle, which persisted for two months, during which time the itching continued to be almost intolerable, and when the part was scratched the spots before alluded to would reappear. About or soon after this time the part began to feel thickened, puckered, and hard, and gradually assumed its present appearance.

On the right thigh, about one inch below Poupart's ligament, and nearer the spine of the pubes than the crest of the ilium, commences this singular appearance of the skin, which more nearly resembles the scar left by a burn than anything else. There is a strip, about one inch broad, nodulated and irregular on its surface, and discolored in a peculiar manner, being partly red, with a predominance of a light brown tint.

This strip of disease proceeds down the thigh, following the course of the sartorius muscle as far as the junction of the upper two thirds with the lower third of the thigh, at which point the most marked discoloration of the skin ceases; but it is found, by examination with the finger, that the same condition of the cellular tissue follows the sartorius to its insertion, and also appears to involve the tendons of the internal hamstring muscles.

In the lower part of the same leg the cellular tissue over the anterior part of the ankle appears to have become involved, and, in particular over the internal malleolus, the integument is firmly attached to the bone.

She has at the present time no peculiar sensation in the affected parts, nor is the use of her leg in walking at all impaired.

Since she has been in the hospital she has taken various medicines, without the slightest perceptible effect.

The next is an instance of the disease in its most aggravated form, reported by Dr. Collingwood. (Model 228, 227, pl. 158^{55}, 158^{45}.)

Elizabeth Alexander, æt. 12, resides at Ellirfield, in Hampshire, where her father follows the occupation of shepherd. She has a comfortable home, plenty of wholesome food, and attends the village school. The following account is given by the gentleman under whose care she has been for some years.

"When I first saw Elizabeth Alexander she was about 4 years old, and was a robust, healthy child, and has been in good health up to the present time. When nine months old, she, whilst crawling about the house near the fireplace, touched a piece of hot iron with the left arm, between the elbow and wrist, which soon healed up, leaving a slight scar, not so large or deep as that produced by vaccination, and to my own knowledge she has had no other burn or scald. When seven years old she had a mild attack of measles, which was so slight that she was not confined to her bed for a day, and perfectly recovered from it. A few months after the measles, she had a white spot appear on her left side, below the breast, about the size of a fourpenny piece, with a brownish, hard, inelastic state of skin, about the size of a five shilling piece, surrounding the white spot, and looking as though the skin had been scorched with hot iron, and I asked the question if such had been the case, and was assured by both mother and child that it was not; and in a few weeks I found the brown part of the skin extending to a large circumference, very much more thickened, puckered, and inelastic, giving no pain on pinching up the skin, or on pressure. About six months after, a similar spot made its appearance on the left shoulder, and from a note I made of the case twelve months after, the following were the appearances then presented.

"The shoulder had been affected for a year and a half.

About a year and a half ago a white spot appeared upon the shoulder, surrounded by a brownish discoloration, just as though it had been touched with a hot iron, not painful or tender to the touch; it has gradually extended itself around the shoulder joint and down the upper third of the arm; the skin is shining, hard, and puckered, like the cicatrix from a burn, and the deltoid and other muscles of the shoulder are so diminished as to leave no appearance of their form; the skin thickened, and apparently adhering to the bone, with considerable loss of power and motion, and contraction of the arm.

"About eighteen months after, the hip (left) became affected exactly in the same manner as the side and shoulder. Two years after this, the right shoulder was the seat of mischief of the same nature as that already existing in the other regions."

From the above account, then, it appears that the disease commenced in the left hypochondriac region, next attacked the left shoulder, then the left hip; up to this time, upwards of four years from the first appearance of the disease, the *right* side was unaffected, while nearly the whole of the *left* side was contracted by it. About a year before her admission the right shoulder became the subject of this singular disease, and, on a careful examination I discovered upon her right thigh a small patch of puckered skin about as large as a sixpence, the right leg and thigh being otherwise free. Of the existence of this small patch the patient was ignorant, which was suggestive of its being the commencement of the disease in a hitherto sound part; but on careful watching, for a period of several weeks, it does not appear that it has increased in size, but rather to have diminished, and the patient affirms that whereas the disease has steadily increased as a whole, individual spots or small patches have made their appearance for a short time and have receded again.

On November 10th, 1852, she was admitted into Guy's Hospital, Lydia 18, under Dr. Addison, when she presented the following appearances. The right shoulder is contracted,

hard, and tuberculated, the muscles are wasted, and a strip of skin, about one inch and a half wide, extending from the back of shoulder to the inner part of the elbow, is bound to the bone. This part was formerly ulcerated, and the only part which ever was so. It now presents a scaly appearance, and is very hard. The left shoulder is more tuberculated, and more hide-bound, but the disease on this side is more confined to the shoulder proper, and does not extend far down the arm. On the front of each shoulder is a considerable patch, but the chest is otherwise free. Both the elbow joints are tightly contracted, and permanently bent at nearly a right angle, and the forearms and hands are considerably wasted. The fingers are nearly all bent inwards, and the hands are small, like those of a child six or seven years old.

From the lower angle of the scapula, a semilunar patch (the original disease) runs round to the mesial line, half way between the umbilicus and the nipples. A large irregular patch exists on the left side, immediately below the umbilicus.

The outside of the left thigh is affected throughout its whole length, together with the whole of the left buttock; the left calf is wasted, and measures two inches in circumference less than the right, while the right thigh measures two inches and three quarters more than the left. The left foot is contracted, and the ankle stiff; the toe is pointed downwards, and she walks upon the ball of the toe.

The right thigh is free from the disease, except a small irregular discoloration about as large as a sixpence, on the front of the thigh. These hard shining places have diminished sensibility, and never were painful. None other of the family ever was affected with the same disease. Her general health is excellent.

Case of Keloid disease. Furnished by John Birkett, Esq., surgeon to Guy's Hospital. (Models 220, 221, pl. 158[56].)

E. K—, æt. 31, a female, was born in Devonshire, lived

some years in the country, but the greater part of her life has been passed in the suburbs of London.

She married at the age of 15 years and 8 months, was confined with her first child at 16 years and 8 months, and never menstruated until after her marriage. She has given birth to eight children, all of whom she suckled with both breasts, although most with the left.

Of regular and temperate habits; she has of late subsisted, since the death of her husband, by working a mangle.

She has always enjoyed good health, with the exception of palpitation of the heart; and her aspect was formerly healthy. At present she is pallid and careworn, from anxiety, and a scanty means of subsistence.

I first saw this patient in July, 1851, through the kindness of Dr. Bossey, of Woolwich, who had watched the case.

In December, 1850, and whilst suckling her last infant, she felt an acute pain under the right arm, and observed a curious appearance in the skin of the part.

Now, July, 1851—six months from the discovery of the disease—it occupies a surface of about six inches by three in extent. It is situated on the axillary half of the right mamma, and extends into the right axilla. The skin feels rigid, as if the tissues were of the nature of parchment. It exhibits a peculiar corrugation, resembling that state of the integuments known as "cutis anserina," in an exaggerated condition. It is of a peculiar dull, yellowish tint, resembling that of ivory. The part is painful; often there is numbness, and at other times sharp, tingling, shooting pains. The right nipple is retracted—more than usual, for it has never been so well developed as the left.

A patch of the same disease, about one inch square, is developed in the skin of the left axilla.

In the summer of 1852, a third patch was developed, in the skin of the inside of the left arm.

At present—and I saw her in January, 1854—the diseased patches of skin have but little changed their appearances.

They have all increased a little, they all give her more or

less pain, and no treatment hitherto adopted has produced any beneficial result.

The patch on the right breast and axilla is longer; the nipple is deeply retracted, indeed invisible, and the gland atrophied. She is much more obese than when I first saw her, and her general health is very good.

The application which seemed to afford her the most relief was the liquor plumbi diacet. dil.

Second Communication.

ON THE

BLOOD AND EFFUSED FLUIDS

IN

GOUT, RHEUMATISM, AND BRIGHT'S DISEASE.

BY

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In February, 1848, I had the honour to communicate to the Society a paper on the condition of the Blood and Urine in Gout, Rheumatism, and Bright's Disease, which appeared in the volume of the 'Transactions' for that year; I now offer one intended as supplementary to the first, and introductory to a third paper, which I hope in a few weeks to bring before your notice.

The principal points established in the first paper having reference to the subject matter of the present communication are as follow:

1. The discovery of uric acid in the blood.

2. Its existence in very minute quantities, mere traces, in healthy human blood, and in that of some of the lower animals, as the duck.

3. Its augmentation in that fluid in certain pathological conditions of the habit.

The mode then recommended for its discovery and estimation was, to extract from carefully dried blood serum, by

means of hot alcohol, such matters as are soluble in that menstruum; then taking up, by hot water, the urate of soda, and after evaporation, either crystallising that salt, or by the addition of a foreign acid, liberating the uric acid, and afterwards collecting and weighing. This process requires considerable time and care in the manipulation, especially if any attempt be made at determining the quantity; and hence, although it is a method most desirable to have recourse to, in investigating the pathology of disease, yet it is one which cannot readily be employed in clinical medicine.

To obviate this difficulty, I have devised another mode of ascertaining the presence of uric acid in the blood, which I have been in the constant habit of using clinically during the last four years, and with the results of which I have every reason to be well satisfied; it is a method which can be readily employed by every medical practitioner, and which has the advantage of requiring for its performance the abstraction of only a very small amount of blood. I have named the process the "Uric Acid Thread" experiment, which is thus performed.

"Take from one to two fluid drachms of the serum of blood, and put it into a flattened glass dish or capsule; those which I prefer are about three inches in diameter, and about one third of an inch deep, which can be readily procured at any glass house; to this is added the strong acetic acid of the London Pharmacopœia, in the proportion of about six minims to each fluid drachm of the serum; a few bubbles of gas are generally evolved at first; when the fluids are well mixed, a very fine thread is introduced, consisting of from one to three ultimate fibres, from a piece of unwashed huckaback or other linen fabric, about one inch in length, which should be depressed by means of a small rod, as a probe or point of a pencil. The glass is then put aside in a moderately warm place, until the serum is quite set and almost dry; the mantelpiece in a room of the ordinary temperature answers very well, the time varying from eighteen to forty-eight hours, depending on the warmth and dryness of the atmosphere.

"Should uric acid be present in the serum in quantities above a certain small amount noticed below, it will crystallise, and during its crystallisation will be attracted to the thread, and assume a form not unlike that presented by stone sugar upon a string (see fig. 1). To observe this appearance, a linear magnifying power of about fifty or sixty, procured with an inch object-glass and low eye-piece, or a single lens of one sixth of an inch focus, answers perfectly. The uric acid is found in the form of rhombs, the size of the crystals varying with the rapidity with which the drying of the serum has been effected."

To ensure perfect success, several precautions are necessary.

1. The glasses should be broad and flat, as above described: watch-glasses of the ordinary kind are not good, being too small, thus allowing the fluid to be frequently spilt; and too much curved, causing the film of partially dried serum to curl up and split.

2. The acetic acid should be neither very strong nor weak. Glacial acid often forms a gelatinous compound with the albumen of the serum, and the appearance of flakes; and very weak acid adds unnecessarily to the bulk of the fluid. By experience I find the acidum aceticum (Pharmacopœia Londinensis) to be well suited for the experiment.

3. The character of the thread and its quantity is of some moment. Very smooth substances, as hairs or fine wire, but imperfectly attract the crystals: if the number or length of the fibres be too great, and the amount of uric acid small, the crystals become much scattered, and therefore but few appear in the field of the microscope. The glass should not be disturbed during the drying of the serum, or the crystals become detached from the thread.

4. Some attention to temperature is necessary; if the serum be evaporated at a high temperature, above 75° Fahr. for example, the drying may take place too rapidly to allow crystallisation; the temperature of an ordinary sitting room answers well for the purpose.

5. If the serum is allowed to dry too much before the examination takes place, the surface becomes covered with a

white efflorescence consisting of phosphates (see fig. 2), which may obscure the thread; this can be removed by the addition of a few drops of water before putting the glass under the microscope; sometimes over-drying causes the serous film to become cracked or fissured throughout, as well as covered with the phosphatic efflorescence.

6. It is well, when practicable, to put up two or more glasses with the same serum.

7. The blood should be recently drawn; that is, no change or decomposition should have been allowed to take place before the experiment is made; the reason for this precaution will be spoken of below.

Delicacy of the above Test for Uric Acid.—The serum of healthy blood, and that of blood from patients suffering from most diseases, gives no indication of the presence of uric acid by the "uric acid thread" experiment; and this absence of very extreme delicacy is of itself a most valuable quality, as only in blood containing an abnormal amount of this principle, will the acid be indicated. In my first communication, where the results of several quantitative determinations of the amount of uric acid in the blood in gout and albuminuria were given, it will be seen, that in 1000 grains of serum it varied usually from 0·045 to 0·175 grain; these numbers were necessarily smaller than the quantities which really existed, being those actually separated and weighed; guided by these results, I have endeavoured to ascertain the value of the "uric acid thread" experiment by the following series of observations. For this purpose I have taken serum of blood from the healthy subject, in which the most careful analysis could with difficulty show the presence even of a trace of uric acid, and to this serum have added the acid in the form of urate of soda in certain definite proportions. After testing such serum in the manner above detailed, the following results were arrived at:

1. Serum with the addition of uric acid in the proportion of 0·010 grain in 1000 grains } gave no indication of uric acid.
2. Serum, containing 0·020 grain in 1000 grains } " no crystals of uric acid.

3.	Serum,	containing	0·0250	grain	in 1000	grains	gave	2 or 3 crystals on thread.
4.	„	„	0·030	„	1000	„	„	a few crystals.
5.	„	„	0·040	„	1000	„	„	several crystals.
6.	„	„	0·050	„	1000	„	„	moderate sprinkling of crystals on thread.
7.	„	„	0·060	„	1000	„	„	thread pretty freely covered with crystals.
8.	„	„	0·080	„	1000	„	„	very numerous crystals on thread.
9.	„	„	0·100	„	1000	„	„	abundance of crystals, more than *usually* found in serum.
10.	„	„	0·200	„	1000	„	„	thread completely covered with crystals of uric acid, and numerous scattered crystals.

It appears, therefore, that an amount of uric acid equal to 0·025 gr. in 1000 grains of serum, in addition to the trace existing in healthy serum, is required to be present in the blood before the "uric acid thread" experiment gives indications of its presence, and hence the appearance of the uric acid on the thread becomes complete evidence of an abnormal or morbid quantity in that fluid.

Changes which Uric Acid undergoes in the Blood when removed from the Body.

In enumerating the precautions which should be observed in making the "uric acid thread" experiment, it was stated that recently drawn blood should be employed, and the importance of this will be seen from the following observations, which at first perplexed me not a little. Having ascertained the presence of uric acid in the blood in many cases, and put aside the serum for a time, it was found that on repeating the experiments, no indication of the

presence of that acid could be discovered; this circumstance more frequently happened in the summer months. On closer examination I found that the serum had usually undergone some slight decomposition, which gave me at once a clue to the explanation of the phenomenon, namely, that uric acid existing in blood is broken up, or undergoes a species of fermentation, when the albuminous portion of the serum becomes altered in character. In order to verify this, the following experiment was repeatedly made, and with uniform results. Uric acid in the form of urate of soda was dissolved in serum in the proportion of from 0·10 gr. to 0·30 gr. to 1000 grains of serum, and the fluid allowed to become putrid. The whole of the acid was found to be destroyed, no indications being afforded by the "uric acid thread" experiment, although at first abundance of crystals were obtained.

I have made some few experiments in order to discover the change which the uric acid undergoes under the above-mentioned circumstances.

When submitted to the action of certain oxidising agents, as the puce-coloured or per-oxide of lead, it is broken up into oxalic acid, urea, and allantoin; and when the oxide is in excess, the oxalic acid is further oxidised and converted into carbonic acid. This fact led me to try whether oxalic acid might not be formed in the blood-serum from a change in the uric acid, and for this purpose I made daily observations on such serum during its decomposition, and found evidence of the formation of oxalic acid in the occurrence of octohedral crystals of oxalate of lime; after a time these crystals appeared to become less numerous, and at last to vanish. I have also evaporated the serum when decomposition was taking place, and treated the residue in the manner described in my paper on 'The occurrence of Oxalic Acid in the Blood,' published in the 32d volume of the 'Medico-Chirurgical Transactions.' Many crystals of oxalate of lime were thus obtained for the most part octohedra, some agglomerated into oval bodies, some similar to dumb-bells. To make the experiment more conclusive, I have taken serum

of blood not containing an appreciable amount of uric acid, divided into two parts, and to one portion have added urate of soda in small amount, and allowed both quantities to decompose; it was found that in the portion of serum to which the urate had been added, oxalate of lime octohedra were formed, but not in that portion free from uric acid. The microscopic examinations were made with object-glasses giving a linear magnifying power of from 200 to 400. Much further investigation is required on this subject; enough, however, has been done to show that the study of these changes is not without interest to the pathologist, for there can be little doubt that oxalic acid is formed in the animal body, not, as formerly supposed, from the oxidation of saccharine matters, but from the decomposition of uric acid. Very many observations on the occurrence of oxalic acid in the blood of man and the lower animals, since the publication of the paper above referred to, have convinced me that such is the case.

Non-occurrence of Uric Acid in the Perspiration of the Gouty Subject.

There are several instances on record in which a whitish powder has been noticed as occurring on the skin of gouty patients, especially after profuse perspirations, and this has not unfrequently been supposed to consist of some combination of uric acid, but no proof of the presence of this body in the excretion from the skin has, I believe, ever been given. In 1853, I adopted the following plan, in order to discover if uric acid is thrown out by the skin of gouty patients. A man was selected suffering from a severe attack of gout, who had been subjected to the disease for a long time, who had many tophi or concretions of urate of soda, and in whom the blood gave, at the time, abundant evidence of containing a large excess of uric acid. Several folds of white bibulous paper were steeped in a very weak solution of potash, and applied for about thirty hours to the abdomen, protected by oil-silk. The papers were rendered acid, and were found to

be strongly impregnated with the perspiration, and to contain much organic matter; these were treated with rectified spirit, and afterwards with hot water, and the watery solution, when evaporated, carefully examined for uric acid. No trace of this body could be discovered, by the murexide test, nor any crystals separated by the addition of acetic acid.

When we consider that the excretion from the skin is very acid in character, and very deficient in saline matters, it would hardly be thought probable that a substance having the properties of uric acid would be excreted with it, either in the free state or that of a saline combination.

Discovery of Uric Acid in certain Morbid Effusions.

I am unacquainted with any published analyses which have demonstrated the presence of uric acid in fluids effused into cavities in disease; but as far back as the year 1848, soon after my first communication to the society, I made some investigations upon this subject, and found indisputable proof of its occurrence. The first of these were made on the abdominal and pericardial fluids in a case of granular kidney, with cirrhosis of the liver, and extensive cardiac disease; for some days prior to death, suppression of urine had supervened.

Abdominal Fluid.—Golden yellow colour, rather thick and turbid; slightly acid in reaction at first, but becoming alkaline on partial evaporation. Odour during evaporation similar to the perspiration. Sp. gravity, 1013·54.

In 1000 parts were contained	Solids . . .	36·40
	Water . . .	963·60
		1000·00

In the 36·40 parts of solids, there were 10·79 parts of albumen, and, on incineration, 5·94 parts of ash were left, the salts consisting of phosphates, chlorides, and sulphates; evidence of uric acid was obtained, in its separation in the

crystalline form, and also of urea; the weights of these bodies, however, were not determined.

Pericardial Fluid.—This was lighter in colour than the above, in other respects similar. Reaction acid. Sp. gravity, 1010·60.

1000 grains gave	Solids	24·53
	Water	975·47
		1000·00

The solids yielded, of—

Albumen	10·53
Salts (ash)	9·70
Uric Acid	0·069
Urea	weight not determined.

Since the time the above analyses were made, I have ascertained the existence of uric acid in effused fluids in several cases where the blood gave evidence of containing an abnormal amount of that principle. I have many times crystallised it from such fluids by the "uric acid thread" experiment.

Discovery of Uric Acid in the fluid artificially effused by the application of Blistering Agents, or in Blister-Serum.

Not unfrequently in practice, for the sake of diagnosis, it is desirable to ascertain the condition of the blood, as to the presence or absence of uric acid, in cases where, from the state of the patient or other causes, the abstraction of that fluid cannot well be effected, and it occurred to me that, probably, the fluid effused by the application of a blister would contain this acid, if the circulating fluid were impregnated with it; experience has proved the truth of this conjecture. The following are some of the results I have obtained by the use of the "uric acid thread" experiment, which may be employed for the discovery of uric acid in blister-serum as well as in blood-serum:

1853. Feb. 9. E. W.	Serum of blood. Sp. gr. 1029·2, at 46° Fahr. Abundance of uric acid on thread.	Feb. 11. E. W.	Serum of blister. Abundance of uric acid.
March 5. J. W.	Serum of blood. Sp. gr. 1026·4, at 51° Fahr. Abundance of uric acid.	March 6. J. W.	Serum of blister. Abundance of uric acid.
March 25. — R.	Serum of blood. Sp. gr. 1028, at 41° Fahr. Abundance of uric acid.	March 28. — R. Attack passing off.	Serum of blister. Sp. gr. 1022·8, at 46° Fahr. Crystals of uric acid.
March 29. J. H.	Serum of blood. Sp. gr. 1029·6, at 47° Fahr. Abundance of uric acid.	April 1. J. H.	Serum of blister. Sp. gr. 1024·8, at 54° Fahr. Abundance of uric acid.
June 30. C. S.	No blood taken.	June 30. C. S.	Serum of blister. Numerous crystals of uric acid.
Nov. 8. M. J.	Serum of blood. Sp. gr. 1026·8, at 62° Fahr. Crystals of uric acid not very numerous.	Nov. 11. M. J.	Serum of blister. Sp. gr. 1024·0, at 65° Fahr. Crystals of uric acid several in number.
1854. January 2. C. F.	Serum of blood. Sp. gr. 1026·8, at 50° Fahr. Abundance of uric acid.	Jan. 13. C. F.	Serum of blister. Moderate amount of uric acid.

It appears, then, from these results, that the fluid effused by the action of a blistering agent applied to the skin, will give evidence of the presence of uric acid when the blood from the same patient exhibits the phenomena, and in the performance of the experiment the same precautions must be taken as have been before indicated when the process with blood-serum was detailed; but, in addition to these, one more circumstance must be attended to, namely, that the application of the blister should not be made to an inflamed part, for it seems that the existence of inflammation[1] has the power of preventing the appearance of

[1] The inflammation attending the production of a blister does not appear to destroy the uric acid.

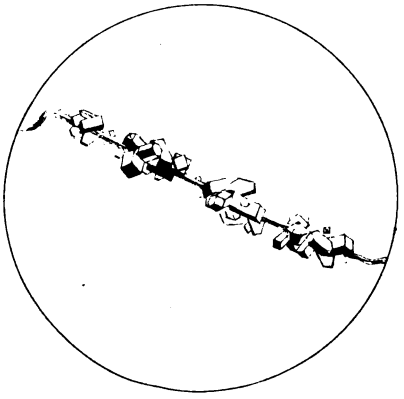
uric acid in the effused serum, as shown by the subjoined results:

1854. January 2. C. F., patient before referred to.	Serum of blood. Abundance of uric acid.	January 2. C. F. ditto.	Serum from blister on inflamed (gouty) dorsum of hand. No trace of uric acid.
1853. Dec. 21. C. C. F.	Serum of blood. Abundance of uric acid.	Dec. 23. C. C. F.	Serum of blister from inflamed (gouty) knee. No trace of uric acid.
1854. Jan. 15. F. P.	Serum of blood. Abundance of uric acid.	Jan. 15. F. P.	Serum from blister to inflamed (gouty) knee. No trace of uric acid.

Should further inquiry confirm the result which the above limited number of observations appear to point to, namely, that during the existence of inflammation in a part, there is a destruction of the uric acid (when such exists) in the blood of that locality, and other independent researches which I have made appear to favour much this idea, it may hereafter throw no small amount of light on the pathology of certain morbid conditions of the system; in the present paper, however, I have abstained, as much as possible, from connecting the condition of the blood with any specific disease, reserving that subject for my next communication on "Gout and Rheumatism," when their differential diagnosis, and the nature of the so-called "rheumatic gout," will be discussed.

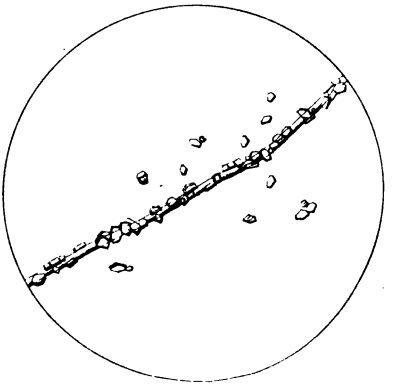
With regard to artificially effused fluids, I may observe that, during crystallisation, the uric acid usually assumes a form slightly different from that in which it occurs in blood-serum, the crystals having a greater tendency to become agglutinated, and form irregular masses, as seen in fig. 3. Lastly, these effused fluids may be employed, not only to ascertain the existence of uric acid, but likewise of other principles, as urea and sugar, which are contained and can be detected in them, when, in the blood of the patient, their presence is capable of demonstration.

Fig 1a.



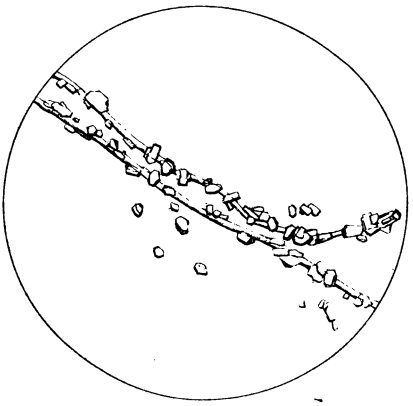
Uric Acid crystallized on fibre from Blood Serum (very slowly dried.)

Fig 1.



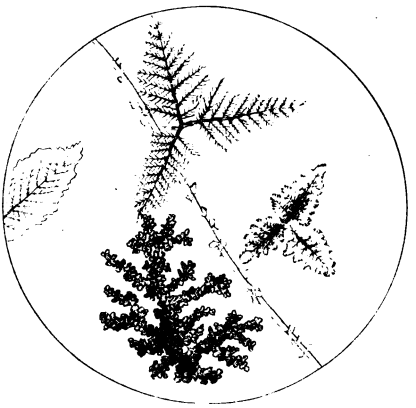
Uric Acid crystallized on fibre from Blood Serum.

Fig 3.



Uric Acid crystallized on fibre from Blister Serum.

Fig 2.



Efflorescence of Phosphates on the surface of the dried Serum partly concealing the Uric Acid on fibre

G.H.Ford Lith

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ON

EXCISION OF THE KNEE-JOINT.

BY

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Whether excision of the knee-joint be a justifiable operation or the reverse, is a point which has been discussed both at medical and surgical societies, and among practitioners at large. There can be no doubt that there exists an extensive prejudice against it, it being condemned by a large majority of British surgeons; but few of the later writers on practical surgery speak of it in a manner to encourage its performance; others are altogether silent on the subject; while in France, if thought of at all, it is so only in connexion with the memory of the Moreaus. This cannot fail to appear extraordinary to those who have given the history of this operation the least attention, for though it must be admitted that in several instances it has terminated fatally, still, as the following facts will prove, few attempts at curative surgery ever promised better at their commencement than this did.

The first well authenticated case in this country (for though Mr. Filkin's, which occurred in 1762, is said to have succeeded, it wants data to substantiate it) was performed in 1781, the cure in this instance being perfect; "the patient was afterwards able to perform all the duties of a seaman." The operation was performed in France in 1792, and certainly with success, for although the patient died three months after of "epidemic dysentery, which, as is well known, carried off the greater portion of those whom it attacked,"

the operator, whose word is above suspicion, states, "I looked upon my patient as cured, for I had no relapse to dread."

Again, in 1823, it was twice performed in Dublin. It is true that in the first case bony union did not take place, but then "disease had proceeded too far; in a word, the case was one to which the operation of excision was not applicable." The patient, however, lived more than three years, in all probability quite as long as she would have done had amputation been resorted to. The second case proved more fortunate; for three years after, the report says, "the patient is able without assistance to stand or walk the length of the day."

In Edinburgh, excision of the knee-joint was performed in 1829, the little patient recovered, so that Mr. Syme, referring afterwards to this case, expresses himself as "having no doubt that ultimately the excised limb will be nearly as useful to him as the other." Mr. Syme repeated the operation the following year, but unsuccessfully, the child dying within the fortnight.

I have brought forward these cases not only as being the *first* in England, France, Ireland, and Scotland, but also to show that an operation which its present advocates are sometimes blamed for performing, was not considered an unsurgical procedure in the hands of such distinguished men as Park, Crampton, Moreau, and Syme, each of whom doubly sanctioned it by its second performance. The result of these cases certainly bears out my previous assertion, that few if any attempts at curative surgery have ever promised better at their commencement; and I may also add that few have ever so soon been allowed to fall into disuse, as from the time of Mr. Filkin's operation until 1850, a period of eighty-eight years, but twelve cases are on record.

In the year above named (1850) this operation was renewed by Mr. Fergusson, of King's College Hospital, and no better proof can be offered of the estimation in which the views of this surgeon are held, together with the determination of many practitioners of the present day to advance conservative surgery to the utmost, than the fact, that in the

space of little more than three years no less than twenty-one operations of excision of the knee-joint are recorded. The subjoined table will show the result of all the cases I have been able to collect, several of which have not yet been published.

EXCISION OF THE KNEE-JOINT, FROM 1762 TO 1854.

Date.	Surgeon.	Name of Patient, &c.	Result.
Aug. 23, 1762	Mr. Filkin	A man, name unknown	Case not well authenticated. It is stated, however (on the authority of Mr. F.'s son), that on Nov. 21 of the same year, "he was got so well, as to require no further attention."
July 2, 1781	Mr. Park	Hector M'Caghen, æt. 33, a sailor	Cured. "Afterwards performed the duties of an able seaman."
June 22, 1789	Mr. Park	Chas. Harrison, æt. 30, a wheelwright	Died, 115 days after the operation, of exhaustion.
Sept. 17, 1792	M. Moreau	M. Claude, æt. 20	Cured. For although the patient died 3½ months after the operation, the surgeon says, "I looked upon my patient as cured, for I had no relapse to dread." The bones had become consolidated.
———	M. Moreau	A man, no name given	Died shortly after the operation.
———, 1811	M. Moreau	A man, no name given	Cured.
Oct. 21, 1809	Mr. Mülder	A pregnant woman	Died of tetanus on the 110th day.
———, 1816	Professor Roux	A man, æt. 32	*Died of* phlebitis on the 18th day.
May 7, 1833	Sir P. Crampton	Susan Connally, æt. 25	Discharged from hospital on 27th June, 1824, "in very good health," but no bony union had taken place between the femur and tibia. Died of phthisis, three years and two months after the operation.
Aug. 4, 1823	Sir P. Crampton	Ann Lynch, æt. 22	Cured.

Date.	Surgeon.	Name of Patient, &c.	Result.
Dec. 7, 1829	Mr. Syme	John Arnot, æt. 8	Cured.
Dec. 28, 1830	Mr. Syme	Ann Mackintosh	Died, eleven days after operation.
July 20, 1850	Mr. Fergusson	A man, æt. 21	Died on the ninth day of acute necrosis of the femur.
Jan. 19, 1851	Mr. Jones	Sarah Hansford, æt. 25	Cured.
April 27, „	Mr. Jones	John Le Gros, æt. 10	Cured.
Sept. 4, „	Mr. Jones	Miss Le Maistre, æt. 30	Died of epidemic dysentery, fourteen days after the operation.
Jan. 25, 1852	Mr. Jones	Robert Quarm, æt. 7	Cured.
May 5, „	Mr. Mackenzie	John Johnston, æt. 28	Cured.
June 7, „	Mr. Page	Wm. Graham, æt. 16	Cured.
Sept. 19 „	Mr. Jones	Ab. Le Feuvre, æt. 19	Cured.
Oct. 30, „	Mr. Fergusson	Ann Goring, æt. 20	Cured.
February 5, 1853	Mr. Mackenzie	Wm. Harrisson, æt. 42	Cured.
March 16, „	Mr. Pritchard	E. H., a man, æt. 20	Cured.
„ 28, „	Mr. E. Thomas	John Harrett, æt. 12	Cured.
April 2, „	Mr. Fergusson	Emma Saville, æt. 28	Died of phlebitis, on the sixteenth day.
——— „	Dr. Steward, Belfast	No name or sex given	Unable to obtain further information than "that the case was encouraging."
April 17, „	Mr. Jones	Wm. Livermore, æt. 12	Cured.
Oct. 31, „	Mr. Gore, Bath	A boy, æt. 14	Recovered.
——— „	Dr. Keith, Aberdeen	A boy, æt. 9	Recovered.
——— „	Mr. Butcher, Dublin	———	Progressing favorably.
Dec. 24, „	Mr. Mackenzie	Miles Christie, æt. 17	Died of exhaustion, the twenty-second day.
Jan. 8, 1854	Mr. E. Thomas	John Christie, æt. 16	Under treatment.
Feb. 15, „	Mr. Erichsen	Wm. Shaw, æt. 7	Under treatment

The above table shows that six of the cases are my own, and their being no longer under treatment, renders them, I apprehend, fit subjects whereby to test the value of the operation. Before proceeding further, however, I must anticipate the very reasonable remark, that my inferences are drawn more particularly from my own practice; the only excuse to be offered is, that previous to visiting the metropolis in November last, I had never witnessed the operation of excision, nor even seen a patient who had submitted to it; and, having been for nearly thirty years deprived of the opportunity of being present when such interesting cases appear, as draw forth the surgical talent of men from whose

practice and remarks so much valuable information is derived, I am forced in advocating excision of the knee-joint to fall back on those cases which have come under my own care, and thus to appear egotistical against my wish; at the same time I must confess my impression, that remarks from one who has been completely thrown on his own resources, are of more value in forwarding the cause on which so much interest has of late been excited, than those of men who are without *practical* experience.

The question may naturally be asked, why this operation has been more successful in my hands than in those of others? The reasons are obvious; consisting in the great advantages arising from the locality and consequent salubrity of the Jersey Hospital. It is not surrounded by high and crowded buildings and a dense population, but has a large piece of ground in front, and a garden at the back, both of which are open to the patients for exercise; and its left wing is scarcely two hundred yards from the sea. The wards are airy, and but rarely crowded; and I have hitherto made it an invariable rule to have a separate, well-ventilated room, as well as a special nurse or nurses, for each of the patients on whom excision or any other important operation is performed. These incalculable advantages are unattainable in metropolitan hospitals, and to them alone, with the stimulating treatment commenced *immediately* after the operation, and steadily persevered in for a considerable time, is my success to be attributed.

The objections raised against the operation are twofold. Its severity, the shock to the system, danger from hæmorrhage, erysipelas, burrowing sinuses, wasting suppuration, &c., forming the first class; and from these it is argued that amputation is much less hazardous. Then, supposing the patient to have overcome or escaped these dangers, we are told that want of union in many cases renders the limb useless; and if the subject be a child, the absence of growth in the excised member is brought forward to prove that a wooden leg, in all instances, is of much greater utility than

the one on which excision has been practised. These once formidable objections can now be combated by existing proofs of their want of weight—the operation having been frequently performed without endangering life further than would have been the case in amputation; and we can from experience affirm that no mechanical contrivance yet known can approach in utility to the limb which has been subjected to this much condemned operation. My own experience enables me fearlessly to assert, that in five of my cases no greater constitutional derangement followed than I have witnessed after the most favorable cases of amputation; in none has the hæmorrhage been sufficient to require either ligature or torsion; nor has the slight appearance of erysipelas, in one or two cases, justified even a moment's uneasiness. It must be admitted that suppuration is greater after excision than it is even in stumps which heal by granulation, in consequence of its longer continuance; but it is, as far as I have been able to judge, less weakening to the system, being much more gradual, and consequently not so exhausting. The first class of objections thus do not appear to be borne out by those cases which have fallen under my observation, and I cannot help believing that the two which were seen last November, by many of the most eminent surgeons in London, must prove satisfactorily that excision in those cases claims a marked superiority over amputation. As regards the remaining objections, I strongly suspect that the case brought forward by Mr. Syme in support of his opinion, will prove the *exception* rather than the *rule*.

Three of my patients were children, under ten when operated on, and in neither of these has growth been stunted, as is apparent from the fact, that the boxes in which the excised limbs were confined immediately after the operation, are now much too short to contain them. The following forcible statement, forming part of the history of Mr. Page's case, with which that gentleman kindly favoured me some time back, goes very far to prove the correctness of my views on each point of the subject.

"I saw the patient this day (January 25th, 1854); he is quite well, the limb is firmly anchylosed and perfectly straight. He has now for some time been employed at the steam looms of a cotton factory, where he works as long as the other hands—and he has to walk or stand the greater part of the day. He walks well without inconvenience or fatigue; in proof of which, he informed me that on Sundays he not unfrequently takes a walk of seven or eight miles. I may mention the important fact, that the boy has grown several inches since the operation, and that both legs happen to have grown equally in length, there being now, as at first, about three inches of difference between them."

The same plan of operation has been followed in all my cases, with the exception of the last: a lateral incision along each side of the joint, and a transverse one immediately over the centre of the patella; the flaps then dissected upwards and downwards, and the patella removed, the joint ends exposed, and so much of the femur and tibia excised as was found in a disorganised state; the bones being then placed in juxtaposition, and secured in a suitable box, similar in some respects to Sir Astley Cooper's fracture-box. This method, as far as I have been able to learn, is the one usually pursued. It had, however, some time before occurred to me that this plan might be improved upon, and having found such to be the case, I can now recommend the latter plan as one possessing the greatest advantages.

It is somewhat remarkable that similar views should, at the same time have been entertained by my friend, Mr. R. J. Mackenzie, Surgeon to the Royal Infirmary, Edinburgh, though I had not then the pleasure of his acquaintance, even as a correspondent. He arrived in Jersey a few days after my last operation had been performed, and on stating to him the method adopted, I found that he had for some time been impressed with its practicability, and probable advantages, and had, moreover, decided to follow it out substantially on the first opportunity, which he did shortly after his return, there being this difference between our

practice—Mr. Mackenzie preserved the patella, but divided its ligament. The subjoined case will show in what respects our operations differ, and also the superiority of this new method over the old one, and will, I trust, induce the greatest contemners of this operation to admit, that at all events in one case, excision of the knee-joint has obtained a triumph in its results which amputation could not possibly have achieved.

My patient, a boy, æt. 12, had for some time suffered under strumous affection of the right knee-joint, which had in no way yielded to the treatment ordinarily pursued in such cases, consequently the operation of excision was performed on the 17th of April last, and in the following manner:—A longitudinal incision, full four inches in extent, was made each side of the knee-joint, midway between the vasti and flexors of the leg; these two cuts were down to the bones, they were connected by a transverse one just over the prominence of the tubercle of the tibia, *care being taken to avoid cutting by this incision the ligamentum patellæ;* the flap thus defined was reflected upwards, the patella and its ligamentum were then freed, and drawn over the internal condyle, and kept there by means of a broad, flat, and turned-up spatula; the joint was thus exposed, and after the synovial capsule had been cut through as far as it could be seen, the leg was forcibly flexed, the crucial ligaments almost breaking in the act, only required a slight touch of the knife to divide them completely; the articular surfaces of the bones were now completely brought to view, and the diseased portions removed by means of suitable saws, the soft parts being kept aside by assistants. In this case the external condyle of the femur was found hollowed out by a large abscess, so that it was necessary to saw off (obliquely) another portion of the carious bone, and to gouge out the remainder, until healthy cancellous tissue was reached, the articular surface of the patella had also to be gouged until sound bone was attained; the bones were now brought in apposition, and the patella and its ligament replaced as

nearly as possible in their natural position, the remaining parts of the operation, together with the after-treatment, were conducted in the same manner as in my other cases.

I shall not enter into details respecting the progress of the case, it is sufficient to say that before the expiration of seven weeks, the little patient was able to turn the limb from side to side easily and quickly, and to raise the leg from the hip upwards without assistance or appliance of any kind; the patella then adhered firmly to the femur and tibia, and its ligament preserved its integrity: unfortunately, however, for some weeks before this gratifying termination occurred, symptoms, which had never before manifested themselves even in the slightest degree, supervened, excruciating pain was felt in the opposite hip, which most energetic measures for a time were unable to mitigate; after many weeks' suffering, the pain by degrees lessened, while the limb became gradually shorter. A spontaneous luxation had taken place, so that at present my little patient when walking, which he does with the assistance of only one stick, presents the following anomalous appearance: on the *right* foot he wears a *thin* shoe, and on the *left* a boot, the heel of which is *upwards of two inches thick;* the existing lameness is only perceptible on the *left* side; and is not apparent on the *right,* and the leg which, under ordinary circumstances, ought to have had at first almost all, and throughout life the proportionably greater part of, the onus, would now be almost the useless member described by the opponents to this operation, without the powerful and almost entire support of the *one on which excision has been performed.*

May not the question now be asked, if in this case amputation had been resorted to, could any patient with a wooden leg on the right side, and a dislocated and diseased hip on the left, be able to walk with no other assistance than one small stick? The answer is too obvious to be dwelt on for a moment.

It is only by comparing cases that we arrive at a right conclusion respecting the superiority of one mode of operating over another; the preservation of the patella and of its ligament is, I feel satisfied, a plan which ought to supersede the other, and be followed out in those cases in which it is practicable; the operation thus performed is rendered more tedious and difficult, but these are secondary considerations when it results in obtaining a more favorable issue.

The rectus acts as a splint, and not only assists materially in keeping the bone in apposition, but also counteracts the natural tendency of the limb to become bent; and I cannot help believing that, should union of the femur and tibia not take place, the preservation of the patella and its ligament must render the limb more useful than it would otherwise be. The following quotation from a paper written nearly fifty years ago, by Dr. James Jeffrey, of Glasgow, is so conclusive on the point that I cannot resist giving it. In speaking of Mr. Parke's and Moreau's operation, this gentleman says—

"It may be said that, though it be an object of importance to preserve the attachment of the extensor muscles in elbow cases, where the joints remain moveable, the surgeon may consult his own convenience at the knee, because that joint, after the operation, is stiff. But it should be considered that, though the crureus and the vasti be extensors of the legs, their auxiliary, the rectus femoris is a flexor of the hip-joint also, and of course a bringer forward of the thigh; and to lose the use of that muscle, in walking, &c., must always be a serious inconvenience, whether the knee-joint be stiff or not; because it acquires power by contraction, the length of the lever with which it acts increasing as the muscle becomes shorter: whereas, most of the other flexors of that joint losé power, their lever decreasing in proportion to the decurtation they suffer in acting. Except, therefore, it be supposed that the ends of the common tendon of the extensor muscles, when cut above the patella, or the ends of the ligament that con-

nects the patella to the tibia, unite after the operation, it is obvious that, by the transverse incision, the power of bringing forward the limb must be impaired."

But, while earnestly recommending the operation of excision as a valuable substitute for amputation, I would not be understood to say, that it can be had recourse to in all cases. In those which are commonly called white swelling of the knee, among others, it may occasionally be quite inadmissible, but in this, as in all other respects, I feel persuaded that the adhering to one mode of treatment, whatever be the circumstances, must produce frequent disappointment; the general features of the case must decide the course to be adopted by the surgeon in this operation, as well as in any other that may come under his notice. As in cases which ultimately necessitate amputation, we are bound in the first place, to exhaust all those means which, if resorted to in an early stage, and judiciously persevered in, may not unfrequently effect a cure; still one important fact must not be lost sight of—the greater the debility of the system before excision, the smaller are our chances of success, while the larger amount of integrity in the soft parts will certainly facilitate the cure. There are some few cases which, though for a time regarded as hopeless, yet under constitutional and local treatment, come to a happy termination; still these cases, while they point out the necessity of due reflection before attempting an operation which may endanger life, must not be too much relied on, and when it is found that constitutional disturbance keeps pace with local symptoms, it appears to me to be consistent with sound surgical principles, that the means of avoiding amputation be no longer delayed; and as in all cases in which excision is decided on, we are, at the same time, prepared to amputate should our diagnosis have proved incorrect, ought we also to be prepared to abandon it altogether, if the admirable plan advocated, and in some instances so successfully followed, by Mr. Gay, that of making free incisions along the joint, offers us the hope that by these means a cure may be

effected? I would, therefore, recommend, as a general rule, free incisions, which can be so made as to constitute the *first* step in the operation of excision, and to allow a sufficient examination of the joint by means of the finger and probe. Should it thus be discovered that the whole joint is in an advanced state of caries, excision may at once be performed, provided the shaft of the femur be not implicated in the disease pervading the joint. The deductions to be drawn from the observations I have been able to make on this subject are: 1st. That excision of the knee-joint is not attended with more danger to life than is amputation. 2d. That while the patient, in the plurality of cases, is much longer under treatment after excision than after amputation, it must not be forgotten, that though a stump may heal much quicker, yet months must often elapse before the parts have attained sufficient firmness to admit of the use of a wooden leg. 3d. That no mechanical contrivance yet known can supersede, or prove as useful as, the limb on which excision has been successfully performed. 4th. That preserving the patella and its ligament, not only ensures greater success, but also gives rise to the question, whether, in those favorable cases where ligaments are not much disorganised, it may not be an object to *encourage* a false joint rather than not. Lastly, that amputation can at any time be performed after the first dangers from excision are passed, as easily and with quite as much hope of success, as after the failure of the remedies ordinarily employed.

Daily recurring facts lead one to believe that the prejudice entertained against excision of the knee-joint is passing away; even while writing this, I have received communications from different parts of the country, proving it to be one of those operations which, however slow its progress has hitherto been, is now evidently advancing. The surgeons of the present day are not satisfied with bare statements, or led away by preconceived opinions, they judge for themselves; and I would humbly hope that my cases may be of some small use in promoting the interests of an operation

which, I consider, not only at present most valuable, but as capable of still further improvements.

I cannot better conclude these imperfect observations than by applying to this particular operation the words used by Moreau when speaking of excisions in general. "It is my wish to show, by the evidence of facts, that the excision of carious joints is, in many cases; a very practicable operation, and one that holds out advantages so unequivocal, that amputation ought to be proscribed in every case where excision may be performed."

ON THE RADICAL CURE

OF

REDUCIBLE INGUINAL HERNIA,

BY

A NEW OPERATION,

WITH CASES AND REMARKS.

BY

T. SPENCER WELLS, F.R.C.S.

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In the year 1847 I assisted Dr. Burmester at Malta to perform an operation for the cure of reducible inguinal hernia. The operation adopted has not been made known in this country so far as I am aware. It was devised by Professor Wützer, of Bonn, and I shall presently describe it.

Dr. Burmester's patient was a gentleman 28 years of age, who had suffered for about eight months from oblique inguinal hernia on the right side. The external ring was dilated, but the intestine had not descended into the scrotum. The inguinal canal readily admitted an ordinary-sized finger. The patient was strong and healthy. He objected very much to wear a truss. No dangerous symptom followed the operation. The patient remained in bed eight days, and was confined to his room a fortnight longer. He afterwards wore a truss for four months. It was then left off, and he had not had any recurrence of the protrusion a few months ago when I heard from him, upwards of six years after the operation.

I have since performed this operation twice myself in two very similar cases; one in the year 1848, and the other in 1850. One patient was a naval officer, the other a groom,

their ages being 18 and 20, and the hernia of recent formation, both oblique inguinal on the right side. Complete success followed, and although both patients are accustomed to very active exercise, no return whatever of the hernia has taken place.

I have not met with other cases suitable for operation in my own practice; but when at Bonn in the year 1850, Professor Wützer showed me two of his patients upon whom he had performed the operation, one only eight days before I saw him, the other about two years before. No unpleasant symptom had followed in the first case, which was going on well, and in the second a radical cure had been effected. In reply to a question I lately addressed to the professor as to the numerical results of his operations he says: "I am not able at present to give you the statistical results of all the cases upon which I have operated, as I have not time to collate them. I can now only say that, since the autumn of 1838, I have repeatedly practised my operation in the Klinik every session before many witnesses, and that I have never seen severe peritonitis follow it, still less any fatal result. All those operated on have not been cured. In several relapse followed, but this was traceable either to the patient's leaving off the truss too soon, or undertaking very hard bodily labour soon after the operation." When at Vienna last year, Professor Sigmund informed me that he had performed the same operation nineteen times in the great hospital of that city, a successful result following in fifteen cases. In two cases gangrene of the integuments followed, and in two others relapse occurred after some weeks, but no death had happened. Professor Rothmund, of Munich, has published the result of his operations on the same plan in the hospital of that capital. He had operated thirty-five times in thirty-two cases, in two years and a half, and no death had followed. His results are almost uniformly successful; but I am informed by a gentleman who wrote to me lately from Munich, that these statements are not deserving of very great weight, as the patients were not watched long after the operation to test the occurrence of relapse. But I trust that a

few remarks upon a method of operating which has led to the results I have just recorded may not prove unacceptable to the Society.

When a surgeon operates skilfully upon a strangulated hernia at the proper moment, he achieves one of the greatest triumphs of our art, for he unquestionably saves the life of the patient without removing or deforming any part of his body. A surgeon who should invent a method of radically curing hernia certainly and safely, would be a great public benefactor, not only by relieving thousands from the inconvenience of wearing a truss, but by averting the danger of strangulation to which they are continually exposed to, in a greater or less degree, through every period of life. It may be said, that the security afforded by a well-fitted truss is almost perfect, and the inconvenience it produces not very great, but patients differ very much in their estimate of the evils of wearing a truss for life; and the frequency with which strangulation occurs among persons who do wear trusses, proves that a more effectual safeguard is required.

Every one must admit that the balance of opinion in the present day among the most experienced surgeons of Great Britain, France, and Germany, is decidedly against any operation for the radical cure of hernia. It must also be admitted, that the opinion has been formed upon facts which fully justify it, for the various modes of operating condemned have been often followed either by death, dangerous peritonitis, gangrene of the soft parts, or by recurrence of the protrusion, so that the patient, after exposure to danger, has been left in no better condition than before. So far as any proceeding implies either opening of the hernial sac, its destruction by the cautery or caustics of any kind, its scarification, the introduction of foreign bodies into it, or the application of a ligature around its neck, every man of sound judgment must agree with the general opinion. For, even if the danger of peritonitis or gangrene were escaped, one would naturally expect that adhesions of the neck of the sac would soon extend, become loose, and give way before the pressure of the viscera, and a new hernial protrusion occur.

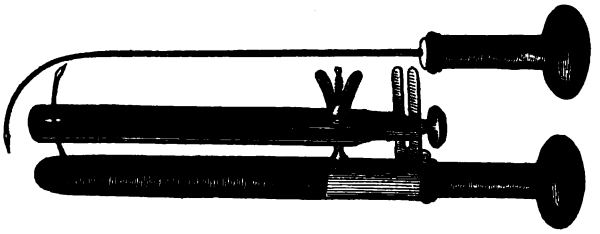
But the question becomes quite a different one when a mild operation of invagination, skilfully practised in properly selected cases, has to be considered. It is true, that Gerdy's method of invagination, although occasionally successful, has led so often to death, or to gangrene of the scrotum and exposure of the testicle, and in the more successful cases has been so often followed by relapse, that it has fallen deservedly into discredit, and is now very seldom performed. But if an array of facts, such as those I have collected at the commencement of this paper, prove that invagination can be so performed as to be safe and generally effectual, perhaps the opinion now entertained may be modified.

The radical cure of reducible hernia can be effected in two ways: 1, by inducing union of the two opposed serous surfaces of the hernial sac by adhesive inflammation and exudation; and 2, by producing close union with an organic body pushed from without into the canal.

I think it better to pass over the various plans which have been adopted with the hope of curing hernia by the first of these plans; for, with the exception of compression, they have all been exploded. A well-fitted truss, properly and permanently applied, gradually excites exudation and adhesion, and in young persons hernia of very considerable size is frequently cured radically without further treatment. But, in adults, compression is only palliative; for the cases in which it effects a radical cure are so rare, that such a happy result is scarcely to be expected in any given case.

The attempts of Belmas, Gerdy, Leroy-d'Etiolles, Signoroni, and Jobert, to close the hernial canal by means of organic union with it of a part of the body of the patient himself, though sometimes successful, have on the whole proved that it is desirable to adopt some plan by which the same end might be attained more safely and securely. It being understood that the inguinal canal must be closed by a portion of skin pushed into it, the first precaution necessary is to effect the closure in such a manner that the adhesive inflammation which it is desired to excite in the hernial sac should not extend to the peritoneal cavity.

Now it has been supposed by Gerdy and others that, when a hernial tumour is pushed before the skin of the scrotum by the finger of the surgeon, the sac is pushed upwards; and that the needle, passed after the method of Gerdy, does not implicate the sac. This, however, is not the case. It is contrary to the anatomical condition of the parts. The serous hernial sac is so firmly adhêrent to the inner surface of the inguinal canal, that it cannot be separated without the assistance of the knife. In one case recorded by Wützer, where a hernia had only existed three days, the adhesion of the sac to surrounding parts by inflammation and exudation was so firm, that separation could only be effected by the knife. If the sac were really moveable, the attempts of Gerdy and every other surgeon to cure reducible hernia radically would necessarily be uncertain, for all we have to trust to is closure of the abnormally dilated canal by adhesive inflammation. We must consider all this fairly in judging of any operation. Professor Wützer did so before he devised the instrument which is now brought before the Society.



He thought that the most safe and promising plan of closing the canal would be by effecting upon its whole inner surface up to the internal ring (and when possible closing the ring itself) an equal mechanical pressure which could at any time be increased or diminished as might be desired or requisite. While keeping a compressing instrument firmly fixed during several days, all use of the knife, and of every caustic under whatever name, should be excluded, and the entrance of air into the peritoneal cavity carefully prevented.

It may be seen that the instrument consists first of a cylinder of very hard wood. This is $3\frac{3}{4}$ inches long, and is made of different diameters, according to the breadth of the canal. It is destined to take the place of the index finger, after the latter has pushed a part of the scrotum through the abdominal ring into the inguinal canal. Towards its anterior blunt extremity it becomes gradually thinner. It contains a canal, lined with metal, which conducts an elastic steel needle, flattened on the point and furnished with a moveable handle. A round opening near the point of the cylinder allows the needle to pass through, so that, when the cylinder has been properly introduced, pressure upon the handle of the needle sends its point along the interior of the cylinder, the skin of the scrotum, the serous coat and coverings of the hernial sac, projecting at last through the integuments. In order to increase the pressure which the wooden body remaining in the canal itself exercises, a moveable case of hard wood is made concave, corresponding to the outer convex side of the cylinder. It is made rather wider than the cylinder, projecting two or three lines on either side, in order to distribute the pressure more equally, and near the end is an opening to receive the projecting point of the needle, which thus fixes one end of the cover over the cylinder. The other end is supported upon a moveable metallic staff; near this is a screw, by means of which cover and cylinder can be pressed together to any degree of strength, so that in a moment the anterior wall of the hernial sac, of the inguinal canal, and the tissues between the cylinder and the cover can be compressed to the precise degree each case may require.

The cylinders are made of various calibres, to adapt them to the different diameters of the inguinal canal, as a great deal depends on the proper filling of the canal by the cylinder, the pressure of which should operate as equally as possible upon all parts of the inner surface of the sac. On the other hand, the diameter of the cylinder must not be too great, or it would be impossible to pass this blunt end to the internal ring, and our object would be defeated; a

diameter of five to seven lines suits such cases. The invaginated scrotum fills the rest of the canal.

The instrument is used in the following manner:—After the hair has been shaved off, the bladder and rectum emptied, the patient lies on his back, with the thighs flexed and raised, and the operator stands or sits between them. The intestine is replaced, if down. The surgeon then places the point of his left forefinger upon the scrotum, about an inch below the abdominal ring, on the affected side, and by carrying the finger, with its palmar surface directed upwards and outwards, through the ring into the canal, he pushes the yielding skin of the scrotum into the canal as deep as practicable; at all events, so far that the apex of the cone of skin thus formed reaches the internal ring. The cylinder having been oiled, it is now introduced by the right hand, withdrawing the finger as the instrument enters. This is not always done without difficulty, and requires some practice: the invaginated skin may return as the finger is withdrawn, and require replacement; or, in cases where the ring is moderately narrow, the cylinder may not be easily introduced by the side of the finger. In this case the finger must be partially withdrawn, to make room for the advance of the end of the instrument. Again, in old herniæ the cellular tissue about the ring is so lax, that the cylinder may be pushed up beneath the skin outside the canal; when this happens, the cylinder is found, on examination, to be much more moveable than when it is within the canal. When convinced that the cylinder properly fills the inguinal canal, the needle is passed through the cylinder, the canal, and the integuments; the wooden cover-plate is placed over it, and pressed against the skin by the screw; the handle of the needle is then unscrewed, and the projecting point is covered by a small piece of cork.

The patient is kept quiet on his back, with the knees bent and supported by pillows. The diet must be so regulated that, on the one hand, we may not prevent a sufficient degree of inflammation in the canal; and, on the other, we may guard against the further extension of it to the perito-

næum. Should symptoms of peritonitis occur, they must be immediately met with energy; but this is very seldom the case if the patient remain quiet. Very gentle pressure should be employed at first; but the screw may be tightened daily, although, in doing this, it is advisable to raise the plate, in order to judge of the degree of existing inflammation, and regulate the after proceedings by it. If this is found to be more than requisite, the pressure is diminished, or the instrument entirely removed.

About the fourth or fifth day the punctured wound begins to suppurate, and there is more or less redness and swelling around it. Professor Wützer says it is not necessary, on the average, to leave it applied more than six days; I have left it seven and eight days. It should be withdrawn as soon as serous fluid, containing fat and epidermis, begins to ooze from the plug; if left longer, injurious suppurative inflammation comes on, and gangrene may commence around the needle puncture. When the instrument is removed, the cavity which remains is filled with soft dry charpie, the puncture is dressed simply, and the whole is supported by a bandage. The patient should remain on his sofa, not only until the cicatrisation of the puncture, but at least eight days longer, so that the fresh adhesions be not broken up by too early movements. In my second case I did not follow Wützer's plan of introducing lint into the cavity, but followed the advice of Rothmund, and endeavoured to procure union of the opposed surfaces of the plug by a graduated compress. No union, however, took place. After either plan, a firm plug is formed in the interior of the inguinal canal, and at first an opening or depression is seen in its mouth; but this entirely disappears after some months, although the plug remains in its place. After a lapse of years, the plug itself becomes gradually diminished, and can scarcely be perceived on examination.

In order to ensure permanent success, the patient should wear a suspensory bandage and a lightly-pressing truss for at least three months after cicatrisation of the puncture; otherwise the adhesions, while fresh and yielding, would be

apt to give way, and the weight of the testicles, if not supported, might tend to draw down the skin of the scrotum to its original position. Powerful exercise of the body should also be forbidden until the truss can be left off.

Two important questions now suggest themselves: 1st. Is the evidence adduced in favour of this operation sufficient ground for admitting it within the province of legitimate surgery? and 2d. If so, in what class of cases should it be performed?

In answer to the first question, I can only refer to the imperfect account of its results with which I commenced this paper. Of 57 cases there reported, no death or dangerous symptom followed in any one, and the proportional success is large. Professor Wützer does not give the number of cases he has treated, but we may presume that it is considerable, and no death has resulted. I have heard that in one case at Brussels death did follow, but that the operation was very unskilfully performed, upon a patient suffering at the time from primary syphilis; so that this misfortune is not to be attributed to the operation, but to the operator. I think, therefore, that if any means are to be resorted to in order to cure hernia radically, that Professor Wützer's operation is that which is the safest hitherto adopted, and the one which offers the greatest chances of success. A truss is always inconvenient: it never gives perfect security against strangulation; it excites in many persons, especially during the first year or two of wearing it, a great deal of mental annoyance, reminding them of a defect in the neighbourhood of the genital organs; it diminishes the aptitude of the wearer for hard bodily labour or active exercise, for there is no truss which can be trusted to keep up a hernia in those who are employed to carry heavy loads, in seamen who have to do duty aloft, or in grooms; yet, as a rule, no warning suffices to keep men from such pursuits because they are ruptured. The cares of a family dependent on labour for support, habit which has grown up from early youth, and ignorance of the dangerous nature of the hernia, are all reasons against an alteration in fixed modes

of life. If all this be taken into consideration, I think that the danger attendant upon such an operation as that I have described, when carefully performed, in fit cases, is slight, when compared with the disadvantages to which a hernial patient is exposed who has to wear a truss for the remainder of life; and therefore that we are bound not to neglect this operation.

As to the class of cases in which it is indicated, perhaps we might say—1st. In all strong, otherwise healthy persons, up to 40 or 45 years of age, who lead a life of active bodily exercise. In such patients where the hernia has only acquired a moderate size, has not become adherent, and where the long diameter of the inguinal canal has not been much shortened by the continued pressure of the intestine, we can most certainly depend upon the excitement of a passive exudative inflammation with subsequent adhesion—in other words, upon a radical cure.

The inguinal canal becomes wider, and at the same time shorter and narrower, the more extensive, old, and neglected the hernia may be. At length the shortness of the canal, the relaxation of its walls, and the large circumference of both rings, form so many impediments to a successful union; and the attempt at a radical cure will be more unsafe in proportion as these impediments increase. All this must be taken into account when considering the second class of cases in which this operation may become advisable, namely, in patients who have not arrived at the age of decrepitude, whose hernial tumours cannot be longer kept up by any mechanical assistance. This may be the case when the inguinal canal is extremely dilated and shortened; in certain species of omental hernia; in cases of great sensibility of the spermatic chord; and in persons with a fat, pendulous abdomen, upon whom the pad of the truss slips when they move. Such persons live in constant danger of strangulation; and in them herniotomy would be infinitely more dangerous than invagination, performed at a proper time. If the neglect have been carried to its highest degree, and the rings have become widely dilated, probably

no method of operation will avail to close them perfectly; yet the patient gains a great deal if, by such a proceeding, the evil can be at least so much diminished that a truss can be successfully worn. In one such case, operated on by Professor Rothmund, in an old woman who had a labial hernia which had reached the knee, four fingers together could be passed through the ring, and no truss was of any use; yet, although no radical cure was effected by the operation, it had the good effect that the hernia could be afterwards properly kept up by a truss.

Allow me to add, in conclusion, that whatever operation be proposed for the radical cure of hernia, it can only be successful when the whole inguinal canal can be so permanently closed by a new, firm, organic substance introduced into it, that, besides the spermatic chord or round ligament, no other parts can find their way through. All methods of cure which can only stop the passage through the abdominal ring must fail in their object, so long as any part of the inguinal canal is left open. In successful cases they may prevent the passage of an inguinal hernia into the scrotum, but are incapable of obstructing renewed protrusion of intestine into the inguinal canal—in other words, of preventing a relapse of the hernia. It is in this respect that Professor Wützer's operation appears to me to be so far superior to others, that I have ventured to bring it before the notice of this Society. It will be for the members to consider how far the evidence I have adduced of its safety and success should lead to its further adoption in this country.

OBSERVATIONS OF MORBID CHANGES

IN THE

MUCOUS MEMBRANE OF THE STOMACH.

BY

DR. HANFIELD JONES.

COMMUNICATED BY

DR. BENCE JONES.

Received April 10th.—Read May 23d, 1854.

It is not at all necessary for the object of this paper to give any detailed description of the structure of the mucous membrane of the stomach. It will be sufficient to refer to the works of Todd and Bowman, and Kölliker, and to state that my own observations are quite corroborative of the accounts they have given. On two or three points, however, a few remarks may be made.

I am inclined to agree with Kölliker that, in the normal condition, there are no glands in the pyloric region of the conglomerate kind or resembling a bunch of grapes. Bruch has stated that he has seen such, and so have I in many cases; but I believe the appearance to depend on a morbid change, in which partial destruction of some tubes takes place, while their remains become convoluted and massed together with adjacent tubes. The low villous prominences which are almost constant in the pyloric region, and occasionally exist in the middle, contain a quantity of nucleated granulous substance, identical with that which is seen in the villi of the intestine. This is liable to abnormal increase, and then spreads as an interstitial formation downward among the tubes. The existence of this nucleated substance beneath

the basement membrane of the intestine (large and small) has not been sufficiently noticed; it must be one of the first seats of morbid change in inflammation, and we have seen bacony matter deposited in it. When I commenced my inquiries into the morbid conditions of the stomach, I was not aware that "lenticular," or solitary glands had been seen in the mucous membrane. Dr. Todd and Mr. Bowman make no mention of them in this situation; Kölliker says, "the lenticular glands certainly do not occur constantly in the stomachs of adults, even if they are possibly always present in those of children, at least in very many cases one meets no trace of them. In others they are seen to be extremely numerous, covering the whole surface of the stomach, yet one can hardly forbid the thought that the diseased conditions of the part, which are always present, have much to do with their formation." From not imagining that they could be normal structures, I termed them simply "nuclear deposits," supposing that they were of new formation. This is, however, in all probability, not generally true, or rather it is true only in a restricted measure. In some animals the solitary glands exist in a very marked manner. If the mucous membrane of a pig's stomach be dissected off, and macerated in dilute muriatic acid, the whole splenic region will show a prodigious number of dead white, round or oval, bodies, the size of a pin's head or a little larger, lying on the deep surface of the mucous membrane, in which they are partly imbedded. These consist of masses of nuclei, with a very little granular matter. In the stomach of the cat they may easily be displayed in the same way, but are much swollen, and lie more completely in the substance of the mucous membrane; they are not confined to the splenic, but are seen in the middle and pyloric regions also. In a rabbit's stomach I could find no trace of solitary glands. In the stomach of a child, æt. 5, who died of a severe burn in a few hours, and whose organs appeared to be all healthy, the glands in question were very numerous. After dissecting off the mucous coat from the muscular, and holding it up before the light, there were seen all

over the surface a great number of minute translucent spots, about the size of a pin's head, in which the mucous membrane appeared to be deficient, but was not apparently depressed. When the mucous membrane was placed in dilute hydrochloric acid, or in tolerably strong acetic, the translucent spots were changed, so as to present a dead whitish opacity. They were most numerous and large in the pyloric region, and were most apparent on the deep surface; in the splenic region they were more numerous than in the mid, and were quite distinct on the inner surface. They consisted almost entirely of masses of aggregated nuclei. In vertical sections these glands were seen lying at the bases of the tubes, and often extending upwards a good way into the substance of the mucous membrane. In a female, æt. 23, single, dying with scrofulous disease and abscess of one ovary, in an extreme state of emaciation, the stomach was found tolerably healthy. On examining the mucous surface, in the way above described, the same translucent spots were observed, in which the tubes were absent, while their place was occupied by nuclei and granular matter. In another female, æt. 19, dying of disease of the brain, set up by mischief in the ear, the stomach, except some mammillation in the mid and pyloric regions, was healthy. In the splenic region there were a great number of minute pin-hole depressions, well seen on looking at the surface by direct light, and appearing as translucent spots with transmitted light. Acetic acid rendered some of these opaque; dilute hydrochloric acid scarcely altered them at all. By microscopic examination it was evident that the tubes were absent in the situation of the spots, which were, in fact, minute cavities containing a few nuclear particles and some oily matter. In the first of these three cases (the child) I think the solitary glands were in some degree abnormally developed. I have not met with them so readily in the stomachs of other children of about the same age. The second case shows the condition in which, I believe, they usually exist in the healthy adult. The third presents them so atrophied as to cause a manifest loss of substance in the wall of the stomach.

It is difficult to fix any exact limit to the healthy development of these glands; all I can say is, that I should regard the gastric tissue as in its most normal and efficient state when there were but few of these glands (or nuclear masses) to be met with, and when those that existed did not encroach materially upon the tubes. It is probable that there are great individual varieties, that they are naturally larger and more numerous in some persons than in others. The idea occurs very forcibly to the mind that these solitary glands, and their groups in the intestine (Peyer's patches), have really no use, and fulfil no function in the human body, but exist in a rudimentary state, in obedience to the law of unity of type. They may almost be regarded as portions of undeveloped embryo substance, existing in inverse ratio to the surrounding specially organised tissues, and with this view their simple nuclear structure, the same that is so common in embryonic parts, is very accordant.

It is, I think, very nearly certain that the epithelial contents of the tubes are thrown off during digestion, and form an important constituent of the gastric juice, probably the so-called pepsin. The evidence for this view is the following:—In some instances the epithelial contents of the tubes do not extend up to the surface, *i. e.*, do not occupy the fossulæ; while in others they are seen fused into an uniform mass, with remarkably definite outline which protrudes from the fossulæ on the surface, and resembles very much a villus or papilla. In one specimen I observed, in a vertical section, a layer of matter, apparently exuded epithelium, covering the surface, which was continuous beneath, with columns of epithelial substance rising out of the fossulæ. Dr. Beaumont seems to have noticed these papilliform protrusions of epithelium in the living organ, as he mentions that, on "applying aliment, or other irritants, to the internal coat of the stomach, and observing the effect through a magnifying glass, innumerable lucid points, and very fine nervous or vascular papillæ, can be seen arising from the villous membrane, and protruding through the mucous coat, from which distils a pure, limpid, colourless, slightly viscid fluid." The

substance of which these papilloid masses are made up is much more homogeneous than the epithelium of the tubes, neither cells nor nuclei can be easily seen in it. The epithelial particles seem to fuse together as they are thrown off. This may serve as an answer to the objection which Kölliker seems to adduce, viz., that the proper cells of the tubes are not to be found at all constantly in the layer of mucus lining the surface. That this is the case I am quite convinced, for, on examining the stomach of a cat killed while digestion was going on, I found, on examining the layer of chyme in immediate contact with the surface, no trace whatever of any cell structure at all, neither of columnar nor spheroidal epithelium. In vertical sections of the mucous membrane there were, however, seen some masses of altered epithelium within the fossulæ, and ready to exude. On the other had, in the stomach of a man who died suddenly after a meal, I found the layer of acid mucus in contact with the surface to consist of abundance of epithelium from the tubes, as well as flakes of columnar particles. Also, in vertical sections, examined without any pressure, the surface was seen to be encrusted with a layer consisting of distinct cells from the tubes. The proceeding which Lehmann successfully adopted in the preparation of an artificial gastric juice, viz., scraping the surface of the mucous membrane with a spatula, and using the expressed matter, indicates pretty clearly that the contents of the tubes are poured out in the formation of the natural secretion. Probably the only difference between different individuals consists in this, that in some the epithelium liquefies completely before it exudes, while in others it exudes as a mass and liquifies more gradually. Though Kölliker doubts that the exuding of the epithelium is a constant and necessary occurrence in digestion, yet he holds that the epithelial contents are all necessary for the formation of gastric juice. I can corroborate the statement of this excellent anatomist, that the acid reaction is much more intense in that part of the stomach where (in the pig) the gastric glandular structure is most developed. This corresponds,

also, with the observation of Messrs. Todd and Bowman, p. 206, vol. II, as to the greater digestive powers of the mid region of the pig's stomach.

The following observation relative to the condition of the gastric mucous tissue at birth, seems worth recording. The stomach of a male infant, who lived only four hours, contained much mucus, of a reddish tint, and markedly acid. In the splenic region the tubes were not distinguishable in vertical sections; they were utterly overlaid and obscured by interstitial nucleated tissue. In the mid region the tubes were rather more distinct; there were numerous large cells of tubular epithelium seen, but the tubes themselves were very much obscured by interstitial nucleated tissue. Acetic acid brought the nuclei into view in great numbers. In the pyloric region the tubes were quite distinct, though there were here also numerous elongated interstitial nuclei. The blood in some of the injected capillaries of the villi was changed into yellow pigment (by the secreted acid). In this instance we have another illustration of the often observed fact, that the embryonic condition resembles very much certain diseased states of adult life. The tissue at a certain part of the ascending scale of development is very like, in its mere morphetic characters, to the same tissue when descending the scale of degeneration.

In the tables accompanying this communication, the following deviations from the typically healthy condition are mentioned:

1. *Nuclear masses;* these, as I have stated, are the solitary glands, and it is doubtful what degree of their development is to be considered as surpassing the physiological limit. It seems probable, both from actual observation, and from the behaviour of the same structures in the intestines, that they may become hypertrophied, and encroach abnormally upon the proper secreting tissue. Again, it is certain that they may undergo atrophy, and thus occasion loss of substance and thinning of the mucous membrane in the spots they occupy. Sometimes their atrophy seems to take

place by a kind of liquefying, so that a cavity is formed containing a clear fluid and some nuclear corpuscles. In other instances there is no distinct cavity, though there may be a depression on the mucous surface, and the mass appears to degenerate fattily, the wasting corpuscles being mingled with molecular oily matter, often in large proportion. From the large, probably hypertrophied masses, there is a gradual transition to the next form of change. It seems worth while to retain the term "nuclear masses," as it expresses correctly the constitution of the so called solitary glands, and, it being clearly understood that they are not actually abnormal structures, separates them in a marked manner from the proper secreting tissue.

2. *Diffused nuclear formation*, in extreme instances, extend uniformly throughout the mucous membrane. The nuclei are mingled with more or less granular matter, and the tubes are more or less atrophied and obscured by the interstitial deposit.

3. *Inter-tubular fibroid formation*, this is very commonly associated with the preceding, and consists simply in this, that the exudation in which the nuclei lie, passes into the form of a more or less fibroid or homogeneo-fibroid stroma. In this, elongated or fibre-forming nuclei may sometimes be seen. The material is very similar to that which thickens the Glissonian sheaths in some cases of cirrhosis. In some cases a change takes place in the tubes themselves, such that they become converted into nucleated substance, similar to that which surrounds them. Their epithelial contents are changed into a granular mass, containing many more nuclei than in the healthy state, while the homogeneous wall of the tube wastes and disappears, and so the intra-tubular nucleated mass blends with the extra-tubular, and the whole mucous membrane is converted into an uniform material loaded with nuclei. In extreme cases the tubes are utterly atrophied, and the whole thickness of the mucous membrane is occupied by fibroid or granular stuff, in which some altered remnants of the tubes may be brought into view by means of acetic acid. The basement membrane of

the surface is often absent in parts where there is much inter-tubular formation, and the nucleated fibroid tissue is then exposed. It may, however, have been covered in by the columnar epithelium during life.

4. The tubes appear, in some instances, *to decay spontaneously*, or, at least, not from the atrophic pressure of new formed fibroid tissue; the mucous membrane may then present a mere mass of granular and celloid débris, with interspersed fat vesicles and fatty matter.

5. *Black pigment* may be deposited in the mucous tissue sometimes in great quantity; it is occasionally within the tubes, more often between them. It appears in the form of granules and masses. In other cases yellow pigment is to be found. Both are to be regarded as proceeding from altered hæmatine.

6. *Cystic formation* is occasionally met with; it seems to take place in three ways: (1.) A nuclear mass liquefies, and leaves a cavity which is occupied by a clear fluid. (2.) While atrophy of the tubes is taking place, a portion of one becomes distended into a cystic cavity. (3.) A cyst is produced (*de novo*) as a large vesicle, a true new formation.

7. *Mammillation* is often seen in lesser degrees, and not unfrequently well marked. It affects especially the pyloric third or half of the stomach. To obtain a good view of it, or indeed not to overlook it, it may be absolutely necessary to wipe off a thickish layer of tenacious adhering mucus. It seems to be of two kinds, or to be produced in two ways. One may be called healthy, and appears to depend on some unusual contraction of the corium of the mucous membrane. That this may take place is very intelligible from the circumstance stated by Middeldorpf, and confirmed by Kölliker and Brücke, that there exist numerous organic muscular fibres in this layer. I have observed that this mammillated appearance is produced in some specimens in a very marked manner, or, if not entirely produced, rendered much more striking by immersing the mucous membrane in water, or in dilute acid, which seems to have a constringing action on some of the component tissues, probably the corium. The

other form of mammillation is morbid, and seems to be essentially connected with fissuring of the mucous membrane, or local atrophy. The thickness of the mucous layer is tolerably uniform in the healthy state, but in some cases when it is dissected off and held up to the light, it is seen to be much thinner in certain parts than elsewhere. The glandular layer seems to be, as it were, broken up into separate portions by fissures running through it. This condition may exist without any mammillation. A section made at right angles to the surface across a depression between two mammillæ shows the tubes in that part shortened, sometimes at the free surface only, sometimes at the deep also. The cause of the shortening seems to be in many instances the disintegration of a superficially seated nuclear deposit. The notching or depression thus produced is sometimes so deep as to fissure the mucous membrane quite down to its corium. In some cases the notching may be the result of simple atrophy, or superficial ulcerations, or such cracks as occur in psoriaris of the skin. The following case is a good example of atrophic change taking place extensively with partial conservation of the healthy structure:—A man, æt. 57, died from a fracture of the skull. The surface in the splenic region at its lower part presented numerous spots about the size of a pea, much more prominent than the intervening surface, and when held up to the light these spots were seen to be much less translucent than the intervals. These prominent spots were more numerous and closer together in the lower part of the mid-region, at the upper part of which, and in the pyloric, there was marked mammillation. The tubes were found to persist, and to be healthy in the prominent parts, while in the intervening thinner they were very much atrophied amid an overwhelming infiltration of nuclei, with circumscribed nuclear deposits at the bases of the tubes. It seems pretty clear that there is a good deal of analogy between morbid mammillation, the result of organic change, and the granular condition of a wasted kidney. The mammillations and the granulations are the parts where most of the natural tissue remains.

8. *Gathering up of the lower parts of the tubes in* the pyloric region so as to form a group of convolutions something like the acini of a conglomerate gland is often observed. It is not quite clear how the change is produced. It seems as if several tubes lost their upper parts by obliteration, and that their then remaining portions were drawn together and convoluted. In an extreme instance the groups of convolutions are found lying beneath the mucous surface, surrounded by fibrous tissue, and manifestly destitute of any outlets. In these cases the epithelial contents of the tubes are commonly fatty and wasted.

9. There is much difficulty in determining exactly what conditions of the *epithelium of the tubes are unhealthy*. Their contents are often of a very opaque fatty aspect, especially in their lower half; but this scarcely seems to be abnormal. In a few instances I have observed an apparently true fatty degeneration of the epithelium, the nuclei and cells being converted into shrunken fatty masses. Not unfrequently the epithelium appears more or less stunted and atrophied, or of a less soft, finely mottled aspect, and its cells look withered and shrunk. In the catarrhal condition it is pretty certain that it is not only the epithelium of the surface and fossulæ (the columnar), which furnishes the abundant mucus, but that of the tubes also, which is thus diverted from its proper use. Large cells from the tubes may not uncommonly be seen imbedded in the tenacious plasma. Sight, however, is quite inadequate to detect the qualitative changes which the epithelium in these and other cases undergo.

10. *Self digestion,* in slighter degrees, is of very common occurrence, and is invariably confined to or most marked in the splenic region. The mucous membrane is stained more or less deeply of a reddish colour, is less thinned, very slippery, difficult to hold so as to make a section, and semi-translucent. The tubes appear in some measure wasted, the submucous white filamentous tissue partly dissolved, and the blood in the vessels converted into yellow pigment. In much rarer cases the mucous membrane is destroyed, all except a slight coating that still remains along some of the vascular

ramifications which are seen coursing as black streaks on the white submucous tissue. The nerves and vessels are seen altered just as when they are treated with strong acetic acid; their nuclei are rendered very apparent.

11. Small dark red circumscribed spots seen on the surface of the mucous membrane are manifestly the result of hemorrhage, or at least of the exudation of hæmatine. The microscope shows in these parts an abundance of dark pigment granules. Sometimes in these spots ulceration is manifestly taking place; the surface is sunk, the basement membrane gone, the tubes quite lost, and replaced by a fibroid tissue infiltrated with yellow pigment. With regard to larger ulcers, such as perforate the walls of the stomach, I have not been able to observe anything to distinguish them from other ulcers, or anything that could account for their origin and progress. The base of the ulcer has appeared of a yellowish-grayish aspect, and some of the substance forming it has shown nothing but a low fibroid tissue, with more or less numerous corpuscles and granular matter, in which lie imbedded fat-cells and remains of vessels. In one instance there were numerous mould filaments in the base of a gastric ulcer, and in another instance in that of a duodenal ulcer; but I do not at all suppose that these had any essential connexion with the lesions. The tissues bordering the ulcer have not presented anything constant or to be specially noticed; sometimes they appear tolerably healthy, sometimes they are diseased in the same way as other distant parts, sometimes they are the seat of blood congestion, but this is not often the case. Ulceration, I believe, is essentially dependent on that which we cannot see; viz., a certain quality of the exudation, and a certain alteration of the nutrition of the tissue affected. It may, I think, be pretty safely asserted that examination of an extending ulcer of the cornea would show no peculiarity that could account for the progressive decay, and absorption of the texture. When separation is taking place, both the aided and unaided eye can see something of the process that is going on, but the destructive action is only

apparent by its results. When we understand the nature of the assimilative power, we shall understand also that of the ulcerative. The following highly interesting case, for which I am indebted to Dr. Bristowe, seems to me to have some bearing on the mode in which ulceration occurs:

"A girl, æt. 12, died at a late period of typhoid fever, from copious intestinal hemorrhage. She was extremely emaciated. There was hepatization and purulent infiltration of a large portion of the left lung. The lower part of the ileum presented numerous ulcers. But the most extensive destruction of mucous membrane existed in the colon, especially in the cæcum and ascending portion. From this part hemorrhage had taken place. The mucous membrane of the stomach had a peculiar appearance. It presented a very considerable number of depressions of a roundish, oval, polygonal, or very irregular shape, the area of which varied between that of a silver penny, and a quarter of that size. They appeared to be produced by atrophy of the mucous and submucous tissues. They were generally somewhat paler than the surrounding healthy membrane, and many were studded with black points, apparently discoloured vessels. The black spots, though most numerous in the depressions, were by no means confined to them. The morbid appearance was observed over nearly the whole stomach, but was deficient for an inch or two near the pylorus, and was perhaps most distinct between the cardiac and pyloric extremities. Not far from the pylorus was an irregular depression of the largest size, having all the characters above described, except that in its centre was a small oval darker-coloured pit in which the mucous membrane appeared to be deficient. It had the appearance of a contracting and imperfectly healed superficial ulcer, and the thinner mucous membrane round it was thrown into delicate scarcely visible folds." In the specimen which Dr. Bristowe kindly sent me the general surface was pale, the margins of the spots were rounded over smoothly, and not sharp cut. The spots were manifestly depressed, and the tissue was more translucent in them than elsewhere. On examination of vertical sections, the tubes

of the mucous membrane were found perfectly healthy; but in the depressions they were destroyed, their place was occupied by mere granular débris and oily matter, and the basement line of the surface was lost. There was no particular change in the submucous tissue. The healthy tubular tissue passed rather abruptly into the disintegrating, and there was no deposit or morbid formation of any kind in the parts affected. It was true and simple disintegration and perishing. No injected vessels were seen by the microscope, nor any pigmentary deposits as from exuded hæmatine. The morbid condition in this case was the result, I believe, of extremely depressed organic power. The nutrition of the gastric mucous membrane, in particular spots, failed, and the tissue passed into a state of decay, it might almost be said, of sloughing. This was not identical with ulceration, but it verged nearly upon it, and had life been prolonged, would doubtless have passed into it; indeed, in the large depression near the pylorus, ulceration seemed actually to have occurred. The case may be regarded as a transitional instance between sloughing and ulceration, and illustrates both processes. Inflammation, it seems certain, had nothing to do with it.

12. The mucus which covers the surface of the stomach in gastric catarrh is generally very tenacious, adheres with remarkable pertinacity to the membrane, is neutral or slightly acid, and consists of an homogeneous-granulous fluid, imbedding very numerous columnar epithelial particles, and often more or less distinct remains of the contents of the tubes. The nuclei of the cells from the tubes persist long after the cells themselves are quite disintegrated, and may be seen in great numbers amid the plasma. They must not be mistaken for mucous corpuscles, which I believe are very rarely present. The columnar particles are more permanent than those from the tubes. Small fragmentary crystals of triple phosphate (as I believe them to be, from their solubility in acid) are very commonly seen in abnormal gastric mucus. The contents of the stomach are often of a dirty chocolate colour; in this case the fluid may be acid or alkaline: it

consists of watery mucous fluid, containing besides epithelial debris and remnants of food, numerous meshes of dark orange pigment: these I suppose to result from effused blood or exuded hæmatine, and to be only a less degree of the black matter which is often vomited in cancerous disease. I have observed torulæ in the mucus of the stomach of a diabetic patient.

The tables accompanying this paper have been drawn up from examination of 100 cases taken just as they presented themselves. This way of proceeding is of course less advantageous for ascertaining the symptoms that attend on diseased states; but it gives, on the other hand, a fairer view of the comparative frequency with which such states occur, and seems on the whole the best to pursue in breaking ground upon a subject which is in a great measure new. I am too well aware of the extreme liability to error which besets all statistical inquiries, to bring forward with anything like implicit confidence the results which seem deducible from these tables; I only produce this as a first effort for the ascertaining of points which will require further and more diversified and abler observation to settle completely.

The proportion of males among the 100 cases is very far above that of females, being 65 : 35, or nearly double. This must be borne in mind in estimating the relative liability of the two sexes to diseases of the stomach.

I will first examine the influence of *age* and *sex*. It appears that out of the 100 cases, there were 28 that might be considered quite healthy, or nearly so. Of these, 15 were males, and 13 females, which indicates a decided less tendency to disease in the female sex.

There were 10 under 10 years of age.
13 „ 20 „
16 „ 30 „
19 „ 40 „
23 „ 50 „

The others ranged from 57 to 74. This result indicates sufficiently a tendency to maintenance of the healthy state

in the early years of life, and also demonstrates that organic change is no necessary attendant upon old age. In case 33 there were numerous sarcinæ in the stomach, and symptoms of their presence were observed during life. In case 43 there was the most extreme vascular congestion, which however appeared to be more of a passive than of an active kind, and to be produced chiefly in consequence of great fluidity of the blood, and venous engorgement. In case 62, though the glandular structure was generally healthy; there was an ulcer with thin edges, at whose base a vessel was seen nearly exposed; the mucous surface was also in a state of catarrh.

In 47 cases the splenic and mid regions of the stomach were either healthy, or not greatly diseased, while the pyloric was generally more or less affected. In a few of these the pyloric was as healthy, or more so, than the other regions, but in the great majority the reverse was the case. Of this group, 29 were males and 18 females, a ratio not very dissimilar to that which exists between the numbers of the sexes. This would indicate that the female sex is as liable as the male to minor degrees of disease. Of this series of cases,—

0	were under	10	years of age.
5	„	20	„
14	„	30	„
22	„	40	„
33	„	50	„
40	„	60	„

While 7 ranged from 62 to 77.

Here, again, age appears to exert a decided predisposing influence to organic change. In 2 cases (53 and 67) there were sarcinæ in the stomach; the latter was in a state of catarrh.

In 11 cases there was a moderate amount of destruction of the tubes. Of these 10 were males, 1 female, an excess on the side of the male sex which must be purely accidental, at least in the degree indicated by the numbers.

1	of these was under the age of	10	years.
2	„ were	„	20 „
3	„	„	30 „
5	„	„	40 „
10	„	„	50 „

In 2 cases (Nos. 49 and 68) there were ulcers. In this group it is very apparent how the liability to disease increases with advancing age.

In 14 cases there was a great amount of destruction of tubes. Among these there were 11 males and 3 females. This result coincides with that obtained in the preceding group respecting the greater immunity of the female sex from organic change of the stomach. The numbers, however, are not sufficiently large to make the evidence conclusive. Of these 14 cases there were—

0	under the age of	20	years.
3	„	30	„
4	„	40	„
5	„	50	„
8	„	60	„
12	„	70	„

One was 70, and one was 90. Here again the influence of advancing years is sufficiently apparent. In one of this group, No. 40, there was cancer of the pylorus.

Among the 100 cases were 6 of more or less decided ulceration, which are reckoned also in other classes with respect to the general state of the mucous membrane. It is rather remarkable that among these none were under 48 years of age. A case of perforating ulcer, which I met with after I had completed the above number, was 52 years of age. Including this one, there are seven cases, the average of whose ages is 59. This was to me an unexpected result, as I had believed, from the authority of others and my own previous observation, that ulceration occurred chiefly in young females. Of the seven cases, five were males, and two females. Rokitansky states "that the disease occurs chiefly at the period of puberty, and very often, particularly

in the female sex, as early as the tenth year." He further states "that it is invariably accompanied by chronic catarrh and blennorrhœa of the gastric mucous membrane;" but this I think is hardly the case in England. I have not noted the existence of catarrh in more than three cases out of the seven, and in one of these it is doubtful whether it was at all marked.

In 16 of the 100 cases, the catarrhal condition was observed, the surface being covered with abnormal mucus in greater or less amount. Of these 10 were males and 6 females.

There were 2 under 20 years of age;
5 „ 30 „
7 „ 40 „
9 „ 50 „
10 „ 60 „
And 4 varying from 64 to 77.

The frequency of catarrh thus increases with advancing age; but the earlier periods of life are by no means exempt.

There were 9 cases in which the patients were known to have drank immoderately, and to these 2 more, subsequently observed, may be added. Of these 11, 1 was healthy; 6 were tolerably healthy, or not diseased in any great degree; in 1 there was a moderate amount of destruction of the tubes; and in 3 this was very great. From this it would appear that the habit of hard drinking has not a very marked effect in inducing degenerative disease of the glandular structure of the stomach. The last case I examined especially bears out this conclusion. The man was only 49 years of age; he had been, as reported, "a drunkard and a very hard liver," in the East Indies, had sunk himself materially in the social scale by his misconduct, and died at last within a hospital mainly from debility. Except considerable hypertrophy of the heart, and a fatty state of the liver, there was no very decided organic disease. The mucous membrane of the stomach was much congested, except in the pyloric region. The splenic and mid regions presented a very tolerably healthy state of their tubular structure. In

the pyloric region the tubes were atrophied and obscured by interstitial nucleated fibroid formation. Just such a condition this was observed in numerous patients whose lives had certainly been very unlike his.

Among the 100, there were 18 cases of marked scrofulous disease, not including instances of tubercular deposit, which were but slight, or obsolete. In 4 of them the gastric structures were healthy. In 10 they were tolerably healthy.

In 2 there was moderate, and in 2 there was great destruction of the tubes. The conclusion is that scrofulous disease, using the term in its widest sense, does not exert any marked influence in the production of organic disease of the gastric gland tissue.

Without reference to microscopic examination, which, had it been possible, would have been most desirable, there are found among the 100 cases, 16 of renal degeneration occurring without marked disease of the liver, and 8 in which both organs were diseased. In the former group there were 3 in which the gland tissue of the stomach was healthy (1, however, of these was in a catarrhal condition, and had an ulcer); 5 were tolerably healthy, 1 being affected with catarrh. In 2 there was moderate destruction of the tubes, 1 of these presented two ulcers and a cicatrix. In 6 there was great destruction of tissue, but 1 of them had attained the advanced age of 90.

Of the second group of 8,—1 was healthy, 3 were tolerably healthy, in 3 there was great destruction of the secreting tubes, and in 1 only moderate.

Taking the two groups together, it appears that in one half the whole number there was decided organic change, while the remainder were tolerably healthy, except that one was ulcerated. This result points certainly, I think, to the existence of a tendency in renal degeneration to be associated with similar change in the stomach. That age is not the real cause of the degeneration in the diseased cases appears from taking the average of the ages in the two sets; in the healthy it is 52, in the diseased 51.

There were 12 cases of heart disease, chiefly dilated hypertrophy. 5 of these coincided with renal and hepatic degene-

ration, 1 with renal degeneration only. Of the 12,—4 were healthy, 3 tolerably healthy, in 2 there was moderate destruction, and in 3 there was great destruction of the stomach-tubes. In 1 case of moderate destruction there were also two ulcers and a cicatrix. The stomach disease coincided with renal and hepatic (one or both) four times, once it did not. From this it appears that heart disease, with its usual attendant of venous engorgement, has probably no great influence in the causation of degeneration of the gland tissue of the stomach. In case 43, where the whole vascular system of the stomach was intensely congested, the tubes appeared tolerably natural.

Among the 100 there are found 7 cases of cancer, and to these may be added 2 more subsequently observed. Of these, 1 was healthy, 5 tolerably healthy, and in 3 there was great destruction of the tubes. In 2 of these cases the pyloric region of the stomach was itself the seat of the cancerous disease. The record of the healthy or degenerated state relates of course to the condition of the remaining mucous membrane. As the greater number of the cases were tolerably healthy (as far as regards the stomach), as in one of the diseased there was coincident degeneration of the liver and kidneys, and as the average of the ages of the diseased is considerably above that of the healthy (59 : 40), it cannot be affirmed that cancerous disease has much potency in inducing degeneration of the gland tissue of the stomach.

In only 3 cases out of the 100 is there mention made of the patient's having suffered from chronic rheumatism or gout. In all of them there existed also renal degeneration, and it is not possible to say whether this or that was the cause of the great destruction of gland tissue which prevailed in 2 of the 3 cases.

There are 2 cases of diabetes, in both which the gastric tissue was tolerably healthy.

I am inclined to hope that the appended tables will furnish a good deal of illustration of diseased states of the stomach, which can scarcely be embodied in formal deduction. To aid the reader in his survey, I add references to the cases

which seem most worth his notice. Instances of great destruction of the secreting tubes: Nos. 2, 5, 8, 19, 29, 40, 44, 59, 63, 69, 76, 90, 92, 93. Instances of ulceration: Nos. 6, 7, 49, 62, 68, 80. Instances of the catarrhal state: Nos. 11, 24, 27, 34, 45, 48, 54, 57, 62, 67, 72, 74, 77, 80, 93, 99. Instances in which scrofulous disease was well marked: Nos. 11, 16, 26, 34, 37, 39, 46, 47, 54, 57, 61, 63, 66, 79, 90, 91, 95, 100. Instances in which renal or renal and hepatic disease existed: Nos. 3, 13, 19, 20, 22, 27, 29, 32, 35, 40, 43, 49, 62, 63, 69, 74, 76, 83, 84, 87, 90, 92, 93, 94. Instances of diabetes: Nos. 14, 79. Instances where cancer existed: Nos. 5, 7, 15, 28, 40, 61, 77. Instances in which the patients had been addicted to drinking: Nos. 3, 4, 5, 19, 26, 68, 80, 82, 93.

With regard to the symptoms by which these morbid states might be expected to declare themselves, it has been matter of great disappointment to me to find that they are so obscure as to be scarcely at all noticed in the records to which I have had access. The following case shows that considerable wasting of the glandular tissue of the stomach may take place without any apparent symptom.

E. G., female, married, æt. 52, had been subject for eight years to epileptic fits, occurring very frequently. In one of these she set her clothes on fire, and was burnt severely. She lingered for rather more than a month, and died. She always had good digestion, never complained of pain in stomach, could eat any kind of meat. Was very strong and well nourished. All the organs appeared healthy except the stomach, on the surface of which were several ecchymosed spots, and the ileum and cæcum, in which were patches of deep red congestion. Microscopic examination showed the tubes in the splenic region tolerably healthy; those in the mid-region were utterly atrophied, and replaced by a fibro-homogeneous stroma, densely loaded with nuclei and granular matter; those in the pyloric region were also extremely wasted, and lost amid fibroid formation.

It is possible that in this case the part of the mucous membrane which retained its healthy structure was able by

increased activity to compensate for that which had perished, and to supply an adequate amount of gastric juice. It is, however, remarkable that so considerable change should have occurred without any local symptoms. This probably depended on the atrophic process having been very gradual. Similar instances of latent, though most serious changes, are met with in other parts—as the cardiac valves, the liver, and kidneys; so that the circumstance is by no means without parallels. In the above case, and in others of the same kind recorded in the tables, I believe the change to have been quite independent of inflammation; but in the following case (for which I am indebted to the kindness of Mr. Ancell), attacks of inflammation seem to have been the efficient cause of the morbid state.

A man died about the age of 50, in a state of atrophy and exhaustion. He had suffered for years from dyspepsia and congestion of the liver. The earlier attacks were of an acute character, and were relieved by blisters; the later were of a more chronic kind. He was several times slightly jaundiced, and his skin at last assumed a permanent dingy, greenish-yellow hue. He was much troubled with sickness. Gentle alterative treatment was of much benefit in the earlier periods of his disease, but latterly nothing did him any good. The autopsy showed some diminution in size of the liver, whose cells were much loaded with yellow pigment; there was some thickening of the capsule. The bile was exceedingly yellow, rather abundant. The kidneys were large, very highly congested, and their capsules very adherent; their tubes contained fibrinous casts, and the epithelium was unhealthy, containing a great deal of oil. Small concretions of carbonate of lime were impacted in the mammellæ. The mucous surface of the stomach was marbled and mottled over about its middle; towards the cul-de-sac it was the seat of punctiform injection if not of extravasation of blood. In the part which was microscopically examined, there was very little trace of the tubular structure, the tissue was completely pervaded by nuclear deposit.

I am satisfied that this stomach was extensively affected

by atrophy of its proper tissue, with interstitial nuclear formation, although, as I had not then directed my attention specially to morbid conditions of this organ, the examination was not so satisfactory as those which I have made recently. By a reference to the groups of different cases given above, it will be seen that the catarrhal state is by no means coincident with destruction of the tubes either in its greater or lesser degree. Now the catarrhal state implies a degree of inflammation of the mucous membrane, but this does not seem to have any marked influence in producing the interstitial deposit which coincides with atrophy of the secreting structure.

In concluding this paper, which I feel is but a first labour in a hitherto little cultivated field, I cannot but remark how strongly the degenerative tendency characterises the disease of the present day. We know not whether it was so in former times, but for ourselves the lesson is plain and clear, that the integrity of the vital force, which we call health, must be carefully cherished if it is to be long preserved. From diminished vital power there is no great step to organic decay; and if the one exists any length of time, there is too much reason to fear that the other is in progress. If the researches I have made do nothing more, they show that degenerative change in one important organ is no unfrequent event, and it requires but a moderate pathological experience to show that the same is true with respect to many other parts. How does it then behove us to look out for and anticipate, as far as possible, these insidious disorganising processes, against which our therapeutic endeavours are often so unavailing!

It is a pleasant duty to acknowledge the very kind assistance I have received while engaged in collecting the observations above recorded from the medical staff of St. George's Hospital, and from my colleagues at St. Mary's. To the curators of the museums at both these institutions I have to offer my best thanks for the many friendly offices they have done me, as well as to Mr. Philliten and Mr. Mushen, resident officers at the Marylebone Infirmary.

Cases of Morbid Changes in the Mucous Membrane of the Stomach.

No.	Name.	Age.	Sex.	History. Disease fatal.	Post-mortem Examination.	Condition of Stomach.
1	E. Caswall.	5	F.	Ill 3 weeks or more with fever; improved; she was approaching convalescence; appetite was good 3 days before death, which occurred in an attack of syncope.	Body emaciated; pale. Portions of both lung consolidated. Kidneys unusually firm.	It was typically healthy in all parts, but quite pale.
2	T. Powell.	62	M.	Labourer.—Regular habits; working hard; health seems to have failed 9 months; at end of which he died in a state of extreme anæmia.	Heart large; walls rather hypertrophied; valves of left side thickened, but efficient. Organs generally healthy.	Tubes of stomach almost wholly destroyed; the mucous membrane reduced to a basement membrane, with a thin substratum of granular matter and indistinct nuclei, below which there is a thick layer of fibroid tissue containing in its deeper parts numerous fat-vesicles.
3	J. Lawrence.	45	M.	Nursery gardener.—Has lived and drank hard at times. Rheumatic fever 10 years ago. Died of fever with pneumonia 2 weeks after admission. Complained latterly of pain and weight at epigastrium, as if nothing passed there.	Right lung in great part consolidated. Pericardium adherent to heart. Liver pale and granular. Kidneys not much wasted, but of granular surface.	Mucous membrane in some parts dark stained, for the most part of a light pale pink. In the discoloured parts there is a deposit of black pigment *in* and *between* the tubes, in other parts there are small deposits of yellow pigment. Splenic region—tubes show a tendency to disintegrate; their epithelium is atrophied. Mid region—tubes tolerably healthy. Pyloric region—tubes obscured and atrophied, by interstitial nucleated fibroid deposit.
	W. Leary.	40	M.	Labourer. — A drinker. While intoxicated injured his hand so that amputation of all the fingers and metacarpal	Heart large and flabby: lining membrane blood-stained; blood fluid. Liver affected with marginal fatty degeneration. Lungs	Mucous surface pale; reaction feebly acid splenic, middle, and pyloric region quite healthy.

No.	Name.	Age.	Sex.	History. Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				bone had to be performed. Tetanus came on. He died on 11th day after admission, and 3rd of tetanus.	congested posteriorly. Kidneys healthy; a cyst the size of a nut on the left.	
5	Jas. Walker.	64	M.	Engaged in business. Not hard worked. Has drank much. Ill 10 months, feeling weak and coughing. Has marked arcus senilis. Digestion good until last 2 months; getting worse. He died with profuse hæmoptysis.	Left lung and heart throughout, especially opposite sixth rib and costal cartilage, where there was an oblong, firm, encephaloid, flattish mass. At the root of this lung there was a mass of cancerously diseased bronchial glands, and the tissue around was soft and broken down as from effused blood. Right lung and other organs healthy. No fatty degeneration of cardiac fibres.	Mucous membrane marbled along the lesser curvature; natural to the eye in the greater part of its extent. Cardiac region—tubes very much atrophied by interstitial formation of nuclei and fibroid tissue, some cystic cavities; corium of mucous membrane thickened and beset with nuclei. Mid region similarly affected, but not quite in the same degree. There appeared to be fattily degenerating nuclei masses, and small fat cells here and there. The basement membrane was perfect. Pyloric region—tubes distinct, though still rather obscured by interstitial formation.
6	M. Stark.	50	F.	Widow, charwoman.—Has had good health generally: 11 children. Admitted suffering with dyspnœa, dropsy, and debility. Urine not albuminous. Died by asthma in 2 days.	Dropsical effusion in pleuræ and peritonæum. Left lung œdematous and emphysematous; right lower lobe nearly consolidated from effusion of bloody fluid. Heart flabby; purpuric spots on its surface; valves healthy; mitral somewhat thickened. Liver slightly granular. Kidneys not wasted; surface smooth; very firm.	Stomach rather large, presented a circular contraction 2½ inches from pylorus. Internal surface pale, rugose, of natural aspect, except at the lesser curvature on the posterior surface, where there were two ulcers, a larger one quite circular nearer the pylorus, and a smaller one oblong nearer the cardia. Their margins were not much thickened, evenly rounded, and devoid of the least vascular injection. There were no adhesions on the serous surface corresponding to the ulcers. There was much highly acid fluid

						in the stomach. The tubes in the splenic and mid region were quite healthy, as also in the pyloric, but they were surrounded in this part with fibroid formation. Some fattily changing nuclei deposits were seen in the corium of the pyloric region.
7	R. Westover.	54	M.	Shoemaker and shopman. —Good health till last 5 years; since then has been ailing more or less in chest and side. Admitted in state of semicoma, with paralysis and some rigidity of left side. Died next day. Slight arcus senilis.	A scirrhous tumour, the size of a fist, in posterior lobe of right hemisphere of brain; serous effusion in ventricle and at base. Left lung partially condensed. Heart healthy looking; fibres of left ventricle in process of degenerating fattily. Numerous adhesions around upper and left portions of stomach. Other viscera healthy.	The cavity of the stomach was contracted, in an hour-glass fashion, by a long puckered cicatrix passing transversely across it, rather nearer the pylorus than the cardia. Contents highly acid. Tubes in splenic and mid regions tolerably healthy; in pyloric exceedingly obscured by nucleated fibroid interstitial formation. Basement membrane in this region was lost, and there was little exuding epithelium.
8	M. Kenny.	50	M.	Jaundiced on admission; abdomen tense and tympanitic. He got very weak; lost appetite. Died on 13th day after admission. Some purpuric spots on cheek. The jaundice appeared 5 months before death; he had no illness before; was never well after.	There was great ascites and much effusion into left pleura. Lungs emphysematous. Heart small; valves rather thickened. Universal chronic thickening of peritonæum, and soft, solid, fibrinous exudation in subserous tissue of recto-vesical fossa. Kidneys small; not manifestly diseased; some hemorrhage into tubes. Hepatic cells loaded with pigment, and numerous collections of green or orange pigment throughout the parenchyma. Ducts com. chol. in some degree obstructed.	Stomach internally of healthy aspect, but marbled near the pylorus. Splenic region—no tubes discernible; they are replaced by nucleated fibroid tissue. Mid region in nearly same state; some nuclear masses at the bases of the tubes. Pyloric region—tubes extremely atrophied in the same way. There was a very little exuding epithelium on the surface. The basement membrane was distinct in some parts, lost in others. The submucous tissue was quite natural.
9	E. Cude.	44	M.	Pelvis crushed by falling timber. He died in about 2 days.	Thoracic and abdominal viscera healthy. Bladder and urethra la-	Splenic and mid region—tubes healthy. Pyloric region is spotted and stained by black

No.	Name.	Age.	Sex.	History. Disease fatal.	Post-mortem Examination.	Condition of Stomach.
					cerated. Both pubic bones fractured.	pigment; the tubes are much wasted, and in some parts are quite lost; there is much interstitial fibroid tissue. The epithelium of the tubes is fatty, and there is much oily matter scattered about them, and even in the corium.
10	G. Chambers.	64	M.	Had good health until 9 months ago; since then difficulty in making water. On admission, retention of urine for 24 hours. Urine drawn off excessively offensive, alkaline, turbid, loaded with triple phosphates and muco-pus. Appetite good. Urine became almost black, opaque, and intolerably fetid. He died in a comatose state, having previously had some pain in abdomen, the bladder being empty.	Body in good condition. Brain and thoracic viscera healthy. Two ulcerated openings at the upper part of the bladder were filled up by the intestines. Peritonitis and purulent exudation. Mucous lining of bladder very congested, and of an olive green hue.	Reaction acid. Splenic region—tubes tolerably healthy in some parts, wasted very much in others; some nuclear deposits at bases of tubes. Mid region covered with tenacious white mucus, consisting of epithelium from the tubes; its surface was pale, and its aspect healthy; tubes very much obscured by nuclear and indistinct granulo-fibroid deposit. Pyloric region—tubes distinct.
11	R. Goulding.	52	M.	Cough 1 year. Admitted with muco-purulent, and bloody expectoration. Legs very œdematous; urine slightly albuminous.	Lungs condensed, and tubercles scattered all about; vomicæ at both apices. Heart healthy. Liver enlarged.	Reaction of mucus acid. Mucous membrane much injected in splenic region, and covered generally with tenacious whitish mucus. Splenic and mid region—tubes healthy; much epithelium exuding on surface. Pyloric region—tubes healthy, but much obscured by interstitial nuclear and granular deposit.
12	D. Ford.	28	M.	Injury to head. Fell into coma, and died during the night.	Fracture of frontal and both parietal bones. Masses of coagulated blood on surface of dura	Splenic region and mid region—tubes very healthy. Pyloric region—tubes tolerably healthy, but much obscured by inter-tubular

XXXVII.					mater. The surface of the brain at several parts was bruised and softened. Much clean fluid in lateral ventricle.	and infra-tubular nuclear deposits, extending quite into the corium.
13	J. Bowen.	25	M.	Ill 14 days with cough and severe pain in right side. Tongue brown and dry; skin cold; extreme depression; breath of fetid and gangrenous odour. Died 1 day after admission.	Body in good condition. Lymph and much sero-purulent fluid in right pleura. Right lung's lower lobe consolidated, infiltrated with lymph and pus, in part gangrenous and broken down. Upper lobe of right and left lung healthy. Heart large, but healthy; fluid and lymph in pericardium. Liver large and coarse. Kidneys very large; mottled.	Reaction faintly acid; surface generally pale, with some patches of injection near pylorus, and some mammillation in the same part. Splenic and mid regions—tubes very healthy, with fibroid formation extending between them at their bases. Pyloric region—tubes greatly obscured throughout by interstitial fibroid thickening.
14	J. Thibel.	29	M.	French exile.—Ill 8 months. Much emaciated. Passed, on admission, 10 pints a day of saccharine urine. Appetite had been very good; had failed much lately. He used to take 170 oz. of milk daily, as well as meat.	Body much emaciated; an abscess, containing ℥j of dark brown and fetid pus in apex of right lung, and several patches of more or less solid cacoplastic deposit. Similar patches in left lung; other viscera healthy. Kidneys natural, but the epithelium of the tubes is very fatty.	Reaction acid. Mucous membrane dead white colour. Splenic region—very much altered by self-digestion. Mid region—tubes perfectly healthy; epithelium fatty, some nuclear deposits. Pyloric region—epithelium wasted, fibroid formation in some parts, and nuclear deposit in others.
15	M. Callaghan.	38	F.	Mammary scirrhus of 1 year's duration. Died by gradual sinking.	Firm encephaloid tubercle-like masses in interior of lungs and beneath pleura; one also in liver.	Reaction slightly acid. Splenic and mid region—tubes healthy, with nuclear formation rather considerable at their bases, and extending up among them. Pyloric region—tubes very distinct, with some interstitial fibroid obscuration.
8 16	E. Parfitt.	20 months.	F.	Ill 5 months off and on; very much emaciated. Symptoms referred to chest.	General tuberculosis. Lungs quite stuffed with tubercles. A few in liver and kidney. Mesenteric glands affected.	Stomach contained food; reaction acid; surface pale. Tubes in every part healthy. Epithelium exuding in great abundance from orifice, and forming a layer on surface.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
17	W. Cox.	46	M.	Fell out of a cart on his head; convulsions came on some hours after; they became continuous, and he died in 4 days.	Body in good condition. Blood extravasated under scalp and in the subarachnoid spaces at the vertex, chiefly on the left side. Surface of hemispheres bruised, and blood extravasated in their substance. Left parietal and temporal bones fractured. Thoracic and abdominal viscera healthy.	Mucous membrane mammillated in every part. No vascular injection. Splenic and mid regions—tubes very healthy; epithelium fatty in lower parts, slightly fibroid thickening at bases and mid, and some nuclear deposits in same part in splenic. Pyloric region—tubes tolerably healthy, much obscured by interstitial fibroid formation, their lower ends forming convoluted groups.
18	E. A. Perren.	17	F.	Rheumatism; pericarditis; great restlessness and distress; sloughs formed on back; death by asthenia in 4 weeks.	Pericardium adherent by soft lymph to heart, externally adhered to ribs and lungs, which were engorged; pleuritic lymph exudation on both sides. Heart healthy, except a fringe of vegetations round mitral orifice.	Reaction acid. Splenic region—tubes healthy. Mid region—tubes not wasted, but much obscured by fibroid formation. Pyloric region in same state. Epithelium of tubes exuding abundantly.
19	J. Edgeson.	32	M.	Gout hereditary in family; has had several attacks. Has not lived at all regularly. General anasarca came on after getting wet. Is a painter and glazier. Urine albuminous, and containing casts. Gout appeared in wrist 3 days before death. Pleurisy and ascites occurred during illness.	Whole body dropsical and exsanguine. Brain pale; of good consistence. Lung œdematous, congested at back; fluid and lymph in pleuræ and in pericardium. Peritoneum contained much bloody fluid and fibrinous adhesions. Kidneys rather small, pale, granular, and mottled.	Splenic region—highly congested; tubes so obscured by fibroid and nuclear formation that they are scarcely to be seen; some are undergoing fatty degeneration. Mid region—tubes in same state; a large nuclear accumulation at the bases of the tubes in one part encroached considerably upon them.
20	N. Sampson.	60	M.	Servant.—Has always lived well. Admitted with fracture of femur; 2 days after delirium tremens appeared; he improved, and went on well, except	Body appeared healthy; 4 oz. of fluid in left pleura; lower lobe of left lung completely hepatized, and whole of right. Heart healthy. Calcareous deposit in aorta and	Reaction acid. Splenic region—tubes tolerably healthy; some large nuclear deposits encroaching on them. Mid region—tubes rather wasted; some encroaching nuclear deposits. Much fat in submucous tissue of

				that no union of the fracture took place. About a month after accident, chest symptoms came on, and he died in 3 days.	other large arteries. Liver healthy. Spleen small and pulpy. Kidneys, surface granular and wasted. Very imperfect formation of callus.	these two regions. Pyloric region—tubes healthy; unobscured.
21	Eliza Baker.	10	F.	Admitted with fever in an advanced stage; ill 14 days; abdomen tense and tender; epigastric pain; slept little. Died in 10 days.	Body emaciated; abdomen distended. Peritonitis with fibrinous and sero-purulent exudation had been set up by the irritation of the mucous coat of the ileum; one of these had perforated the intestinal wall, but adhesions had prevented the escape of contents. Abdominal viscera healthy. Lungs congested; middle and lower parts of right consolidated and softened. Heart healthy. Blood everywhere very fluid.	Reaction acid. Splenic region—there was a good deal of sub-tubular nuclear deposit, which in one specimen was so considerable that it formed a layer nearly as thick as the remaining depth of the tubes on which it had encroached. Mid region—tubes healthy; slight fibroid formation encroaching on their bases. Pyloric region—tubes remarkably healthy, and unobscured.
22	H. Boyce.	35	M.	Never had rheumatic fever. Ill, more or less, 18 years with palpitation of heart; more last year; unable to do his work as a costermonger. Anasarca; orthopnœa; heart greatly enlarged; systolic bruit in left side; impulse forcible. Pulse rather weak and unequal. Lungs engorged; absolute dulness in lower half of left back. Urine highly albuminous; contains casts, glomeruli, blood globules. Died in a few days.	Left ventricle of heart very much hypertrophied. Mitral curtains and cordæ tendin. thickened; orifice contracted. Aortic valves thickened. Surface of aorta rough and puckered by semi-cartilaginous patches. Kidneys diseased, enlarged, and mottled. Liver firm; nutmeg-cirrhotic. Lungs engorged; lower lobe of left condensed by Oiss of fluid in pleura.	Mucous membrane appeared healthy, rather more pinky than natural. Splenic region—tubes tolerably healthy; some fibroid formation at their bases. Mid region—tubes very much obscured by fibroid deposit and much wasted. Pyloric region—tubes not much wasted, but obscured a good deal by interstitial fibroid formation. Basement line perfect in all the regions. Very little exuding epithelium. Abundance of fat in sub-mucous tissue.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
23	L. Gunden.	5	F.	Ill 6 months with cold and cough. Palpitation 2 months ago. Three days ago had a fit; after which she had hemiplegia of the right side, and speech was affected. This was not of long duration. There was a loud systolic mitral bruit. She improved in 2 months very much, and was about to be discharged, but got an attack of severe pain in cardiac region, with roseolar eruption on face, and sonorous râles in left chest. Urine was soon after slightly albuminous; œdema appeared, and the face was much swollen at the time of her death.	Body much emaciated. Heart very much enlarged and dilated; filled with enormous black clots. Some pericardial adhesions. Large rough vegetations on mitral flaps; aortic valves healthy. Left lung much condensed; contained traces of recent and older extravasations. Right lung condensed in some degree, and presenting some small cavities filled with puriform fluid. Liver enlarged; very firm; rather dark. Spleen large, dark, firm, with a fibroid nodule in its anterior margin. Kidneys tolerably healthy; fibroid formation was taking place in them.	Reaction acid contained digesting food. Splenic and mid regions; in both, the epithelium of the tubes is very fatty at their deeper parts; no wasting; no intertubular deposit. Pyloric region, tubes very tolerably healthy, but there were some nuclear deposits, mingled with oily matter, advancing upwards from the corium and causing local wasting. Epithelium exuding very abundantly on surface.
24	Jas. Mortimer.	7	M.	Admitted with extensive strumous disease of left elbow and hand, and right ankle. Five weeks after, an attack of violent diarrhœa. Twenty-four days after, urine was of a dark olive green, and highly albuminous; abdomen distended. In 16 days after, he died; having passed latterly very little urine.	Marked pallor; general anasarca. Right lung, upper lobe hepatized incompletely; lower lobe healthy. Left lung healthy; no tubercles in either. Bronchial glands enlarged by scrofulous deposit. Kidneys enlarged; surfaces very white and smooth. Liver large; tolerably healthy. Spleen much enlarged; contained deposits of bacony matter. Some sero-purulent fluid in pelvis.	Reaction acid; surface pale, covered with much mucus. Splenic region—tubes not much wasted, but imbedded in fibroid tissue. Nuclear deposits very marked encroaching on the tubes; epithelium abundant. Mid region in nearly the same state as splenic; nuclear deposits in the substance of the mucous membrane. Pyloric region—tubes not materially wasted, but obscured by much interstitial deposit; nuclear masses exist at their bases, encroaching on the tubes. Fragments of bacony matter seen in the tissue.

25	— Scales.	9	M.	Died with acute desquamative nephritis after scarlatina. No stomach symptoms.	General œdema. Fluid in pericardium. Lungs œdematous.	Reaction slightly acid. Splenic and mid-regions—tubes perfectly healthy; epithelium very fatty. Pyloric region—tubes healthy, containing a less fatty epithelium.
26	M. Haley.	38	M.	Always a hard drinker; much exposed to weather; subject to winter cough; much emaciation; worse last 5 weeks; no hæmoptysis. He soon sank and died.	Tubercles and vomicæ in both lungs. Heart healthy. Kidneys of normal size, dark, and congested. White pericardial patch.	Reaction acid. Appearance healthy, smelling of brandy. Splenic region altered in some measure by self-digestion; tubes natural. Mid-region—mammillated tubes natural. Pyloric region—slightly mammillated; tubes very much obscured by fibroid formation. Fatty degeneration of their epithelium in many parts.
27	J. Macreath.	49	M.	Twenty-four years ago had a paralytic stroke; lately, 2 fits. Has pains all over head; increased greatly by coughing; has a continual noise in his head the last 6 weeks. There is a large carbuncle on the side of face. He sank into coma and died.	Brain healthy. Lungs very œdematous; right rather condensed and softened. Heart healthy; several pericardial adhesions. Adhesions on surface of liver; its tissue soft; highly congested. Both kidneys excessively wasted.	Splenic region somewhat altered by self-digestion; tubes tolerably healthy, but there is a good deal of nuclear and fibroid formation in the sub-tubular tissue, and extending up between the lower parts of the tubes. Mid region in similar state. Pyloric region similarly affected, and the lower parts of the tubes thrown together into convoluted groups. There was much mucus on the surface in all the three regions.
28	M. A. Little.	70	F.	Was extremely exhausted from previous illness and old age. Diarrhœa at intervals during last month. Abdomen became tympanitic and painful. Two days after, she sank.	Body pale and emaciated. Much bloody fluid in both pleuræ. Lungs emphysematous. Heart healthy. Serous and fibrinous effusion in peritonæum. Twenty-five gall-stones in gall-bladder, and much dark black bile. Liver contained several masses of hard encephaloid. Spleen and kidneys healthy. Cancerous stricture of rectum.	Aspect healthy. Reaction acid, rugæ marked, empty. Splenic region slightly mammillated; tubes tolerably healthy; some fibroid formation at their bases. Mid region—tubes healthy. Pyloric region—tubes not materially wasted; lower ends convoluted; much intertubular fibroid formation.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
29	Chas. Hawell.	63	M.	Of rather full habit. Face and neck purple from congestion. General dropsy, dyspnœa, and cough for 1 month. Heart's action irregular. Pulse remarkably feeble and irregular. Expectoration rusty and adhesive. Urine scanty, loaded, slightly albuminous. Died 15 days after admission.	General anasarca; much serous effusion in left pleura, compressing left lung, which was healthy, as well as the right; a small pericardial patch. Heart's cavities all dilated; left ventricle hypertrophied. Considerable calcareous deposit in mitral valve. Much fluid in peritonæum. Liver small; its capsule contracted in many parts. Spleen small; firm; capsule opaque. Kidneys congested; firm; capsules adherent, surfaces granular and cysted.	Strongly acid chyme in stomach. Splenic region—mucous membrance dark-coloured, thinned; tubes very much disintegrated; a prodigious amount of fibroid formation in sub-tubular tissue and corium, which is all full of elongated and fibre-forming nuclei. Mid region—tubes more or less completely broken up; their debris mingled with melanic granules. Fibroid formation as in splenic region. Pyloric region—tubes tolerably healthy, involved in much fibroid thickening; lower ends rather convoluted, and epithelium fatty. Muscular coat greatly thickened near pylorus. Submucous tissue not indurated. No congestion in any part of stomach.
30	G. Morrison	45	M.	A footman. Was exposed to the cold outside a carriage while heated by walking. Diffuse cellular inflammation came on, affecting the right side of the neck, the cheek, and extending down into the axilla. Died by asthenia in 3 days.	Lungs rather engorged posteriorly; other viscera of chest and abdomen healthy, except a few cysts on the kidneys.	Rugæ well marked; no general congestion; but intense injection of the vessels in the margins of some of the rugæ. Splenic region—tubes very healthy. Some nuclear deposits encroaching on the bases of the tubes. Mid region—tubes healthy, but with nuclear and fibroid formation encroaching on their bases. Pyloric region—tubes very much obscured by fibroid formation; here and there the lower parts convoluted. Mid and pyloric regions markedly mammillated; the mucous membrane is thinner in the furrows than elsewhere, and the tubes in the same parts are shortened.

31	E. Wilkins.	29	F.	Fell out of a 3-story window which she was cleaning. Enormous effusion of blood under scalp; perfect unconsciousness; pupils immoveable. She sank and died the same night.	Body well formed; old cicatrices on legs. Blood effused beneath the arachnoid and in its cavity. Inferior part of right hemisphere much bruised on surface. Cranium fractured; bones not depressed. Lungs greatly congested. Anterior part of spleen slightly bruised. Viscera otherwise normal.	Reaction acid. No trace of congestion. Slight mammillation in cardiac and pyloric regions. Splenic region—tubes very healthy, epithelium fatty. Mid region—tubes show a tendency to disintegrate, nuclear deposits and fibroid thickening at their bases. Pyloric region—tubes healthy.
32	R. Anber.	27	M.	Admitted with dropsy of 3 weeks' standing. Had rheumatic fever 12 years ago; no palpitation since. Complexion pale and waxy. Urine smoky; highly albuminous. Had pain in back. The dropsy increased, and he died in about 7 weeks.	Considerable œdema of lower limbs and scrotum. Some serum in both pleuræ and peritonæum. Left lung rather compressed; right œdematous at back part. Heart healthy. Liver weighed 6½ lbs.; its edges much rounded. Capsule of spleen thick and opaque. Kidneys enlarged; surfaces smooth and mottled.	Surface covered with acid chocolate fluid. Splenic region—tubes tolerably healthy. Mid region same, with some nuclear and fibroid formation at bases of tubes. Pyloric region—tubes very much obscured by fibroid formation. Mucous membrane somewhat acted on and altered by acid contents.
33	E. A. Smith.	13	F.	Was quite well until 3 months ago; since when her feet and legs began to swell, and were so 14 days. Pain and palpitation occurred after anasarca had ceased. Much loss of flesh; appetite good. Loud systolic mitral bruit. Cough and bloody mucous expectoration. She improved a good deal, but in about 6 weeks had an attack of bilious vomiting with weight and pain at chest after food. This subsided in great measure under treatment. But she sank gradually, and died about 3 months after admission. Urine was very albuminous at the last.	Body universally anasarcous; very pale. Patches of great congestion and of extravasation of blood in right lung. Left less congested; much compressed by the greatly enlarged heart. Pericardium adherent. Mitral valve apparently efficient; other valves healthy. Kidneys healthy. Liver gorged with blood.	A large quantity of grumous dark matter (altered blood) lining the surface; it contained very much yellow granulous pigment, and numerous sarcinæ. There was emphysema of the sub-mucous tissue. In all the three regions the tubes were tolerably healthy.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
34	L. Famin.	16	F.	Has lost much flesh; was well and strong 3 months ago. Has latterly felt weak, and out of health. Much pain in abdomen. Bowels relaxed; night sweats; cough dry. Abdomen hard, tense, tender. Signs of softening tubercle in the apices of lungs. Appetite bad; slept badly. Sank gradually.	Body emaciated; extremities anasarcous. Very numerous miliary tubercles in lungs. Bronchial glands tuberculous. Heart healthy. Peritonæum thickened; scrofulous deposit in it, and in omentum and mesenteric glands. Intestines vascular and adherent to each other by means of scrofulous deposit. Mucous lining of ileum at lower part ulcerated. Some small tubercles in kidneys and spleen.	Reaction acid. Mammillation of mid and pyloric regions. Splenic region contained numerous small ulcers, the size of a pin's head. No marked injection anywhere. Splenic region—tubes tolerably healthy, but the continuity of their line was often interrupted, either by complete excavations or by wasting of the tubes on their free or their deep ends. There were nuclear deposits in the sub-tubular tissue and in the mucous membrane. Mid region—tissue in nearly the same state; a cyst was seen in one nuclear deposit at the free surface. Pyloric region—tubes much obscured by fibroid formation. The mid and pyloric regions were covered with yellowish green mucus.
35	G. Clark.	55	F.	Admitted moribund; having suffered for some time from cough and dyspnœa, with general anasarca of lower limbs for about 10 weeks.	Body well made; general dropsy. Lungs œdematous, and lower parts softened; right pleura almost full of yellow fluid. Old pericardial adhesions, and local thickening of visceral layer. Both ventricles of heart greatly dilated. Mitral valve somewhat thickened and rigid at its margin. Aortic valves abnormally thin. Much fluid in peritonæum. Kidneys very dark, hard, congested, and cysted. Liver very dark, nutmeg appearance. Spleen dark and very firm, with an opaque, hardened capsule.	Mucous surface injected much; mammillated; lined with bloody mucus; not acid. Splenic region—tubes tolerably healthy. Mid region—tubes tolerably healthy; black pigment in the tops of the short villi; a cyst seen lying near the surface. Pyloric region—tubes much obscured; not wasted. Epithelium exuded abundantly from tubes in mid-region; it consisted chiefly of finely-granulous matter, free nuclei, and small cell particles.

36	H. Jacobs.	37	M.	He had an apparently old spinal curvature; suffering severely from dyspnœa, with cough and difficult expectoration. Chest full and resonant; ribs not moved in inspiration; loud, prolonged expiratory rhonchi. Was much relieved by copious cupping: but in a few days the dyspnœa returned and he died.	No disease of vertebræ; only a considerable anterior posterior curvature. Both lungs very emphysematous in front; bronchi congested, and loaded with muco-pus. Heart large; valves tolerably healthy. Kidneys congested; other viscera healthy.	Mucous membrane of a reddish colour, lined by a layer of dark chocolate-coloured mucus in its splenic half, and by a paler mucus in pyloric half. Reaction highly acid. Splenic region—mucous membrane thin; dark-stained, semi-translucent, and soft; tubes do not appear wasted, but altered by self-digestion. Mid region—tubes healthy, but rather altered by acid. Pyloric region—tubes healthy, unaltered.
37	W. Oliver.	30	M.	Admitted with pretty severe hæmoptysis of 1 week's duration. Had had before similar, but less severe attacks. Had had cough a long time, was much emaciated and very weak. Sputa became in 9 days purulent and free from blood. There were signs of tubercular disease at both apices. He took meat at first, but afterwards his appetite failed. He sank and died in 6 weeks after admission.	Both lungs contained tubercles and vomicæ; a large cavity in the left apex was lined by a smooth membrane. Muco-purulent fluid in bronchi. White pericardial patches. Heart healthy, abdominal viscera also.	Mucous membrane slightly injected; lined with yellowish mucous fluid; acid. Tubes quite healthy in all three regions; epithelium rather fatty in splenic; some inter-tubular and sub-tubular nuclear formation in pyloric.
38	J. White.	42	M.	A dark-haired, robust, muscular man; not fat. Had stricture, for which he was operated on by perineal incision. He died in 6 days, from diffuse cellular inflammation and slight peritonitis.	Much hypostatic congestion in back part of both lungs; they were otherwise healthy, as well as all the other viscera.	Surface smooth; pale. Some congestion in splenic region. The tubes of this part are very tolerably healthy, but altered by self-digestion. Mid region—tubes indistinct: in many spots there are nuclear deposits which are most considerable in the deeper parts of the mucous tissue, but which extend sometimes quite through it,

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
						destroying the involved tubes; in some parts the whole tubular tissue is completely pervaded by the nuclear deposit, and the tubes quite atrophied. Pyloric region—aspect uneven; tubes completely atrophied, black, wasted, and imbedded in a mass of fibroid tissue; basement membrane quite lost.
39	J. Goodacre.	34	M.	Ill 4 months with catarrh, and a sediment in his urine; pain in passing water, and pain in back. Has not lived hard. Urine contained much albumen and pus. He was very low, and could give no satisfactory account of himself.	Heart healthy. Lungs full of tubercles and cavities. Liver and intestines appeared healthy. Long bands of adhesion between the left lobe of the liver and the colon, and the opposite wall of the abdomen. Left kidney riddled by suppurating scrofulous deposits; some deposits of same kind in cortex of right. Scrofulous ulceration of left ureter. Miliary tubercles and extensive ulceration of the mucous membrane of the bladder. Much emaciation.	Mucous surface pale throughout. Splenic region—tubes tolerably healthy, but in some measure altered by self-digestion. Mid region—tubes tolerably healthy, but obscured by intestitial fibroid formation. Pyloric region—tubes very much wasted, scarce seen at all amid the great quantity of fibroid formation pervading the mucous tissue.
40	J. Haylis.	63	M.	Coachman.–Injured his left side 12 months ago; abscess formed just below the left costal cartilages at end of 6 months. He lost appetite; had much indigestion; got much thinner. In 5 weeks he returned to work, but another abscess formed. On admission there was a hard, brawny swelling over carti-	Lower limbs œdematous. Lungs œdematous, otherwise healthy. Peritonæum opaque and thickened. Several large cysts in left kidney; right not of healthy appearance. Liver hard, thickened, and contracted. Old adhesions and induration round liver and spleen. Two or three sloughy fistulous openings in skin of epigastrium led to and opened into a large sloughy	Cardiac portion pale; mid region blackish, discolored; pyloric third occupied by a large scirrhous growth, which formed a sloughing surface covered with fungous growths. Splenic region — tubes tolerably healthy, but tending to disintegrate, and encroached upon by fibroid formation. Mid region—tubes quite gone; tissue thoroughly infiltrated with nuclei and granular matter, fatty here and there. Some groups of pale vesicles were seen in the

				lages of false ribs and region of stomach, and 2 external fistulous openings. Appetite bad; can take no meat; has flatulence. A fecal smell was perceived from the ulcerated part. He sank, and died in about 1 month.	"foyer," communicating both with the transverse colon and stomach. Anterior wall of stomach in one third of it pyloric region destroyed by malignant ulceration. Cortical tubes of kidneys very much broken up; numerous cysts in medullary.	mucous tissue, and not far from the free surface. Basement membrane remained. Muscular coat was thickened, not the submucous. The cancerous mass consisted of fibres externally, and of villi formed chiefly by columnar epithelium internally.
41	Caroline Austen.	6	F.	Died, apparently from asthenia after scarlatina; was ill rather more than 3 weeks. Urine highly albuminous. Some diarrhœa occurred, and bed-sores formed.	Body pale; thin. Lungs exhibit in numerous parts patches of a pale colour, somewhat elevated and indurated; in other parts there are patches of dark congestion; some of the indurated patches contain puriform matter. Kidneys large and flabby; cortical tubes obscured by interstitial fibroid formation; some of them healthy; epithelium of others wasted, and tube dilated.	Stomach empty. Reaction not acid. The mucous membrane in all three regions was perfectly healthy; tubes quite natural.
42	H. Newt.	42	M.	6 or 7 weeks before his admission he had arrived from Egypt; had good health while abroad, except that on two occasions he had attacks of pain and gnawing sensations at epigastrium, with diarrhœa. Was confined to bed 3 weeks before admitted with cough, expectoration, pain in right side, and short breath; was much emaciated; there were five irregular crepitations	Body in tolerably good condition. Some fluid and fibrinous exudation in pericardium; white patch on heart, which was healthy. Left lung œdematous; right compressed by copious pleural effusion; a few tubercles in its apex. Some red fluid in peritonæum. Liver very large and adherent to diaphragm: it contained numerous masses of fibrine in various stages of softening; some of them were very large; one in the right lobe	Stomach dilated, except near pylorus; it contained a quantity of acid reddish fluid. Splenic region—mucous membrane exceedingly softened, quite translucent; tubes visible, but appearing as if half dissolved and reduced to faint shadowy streaks, containing opaque dots; their number did not seem diminished; no other trace of disease. Mid-region—tubes quite healthy. Pyloric region—tubes very tolerably healthy, but show some tendency to disintegrate; and there is some nuclear and fibroid formation encroaching on their bases, sometimes considerably.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				throughout both lungs. In about 2 weeks he brought up a large quantity of thick pus, as from a cavity; was very low, and had diarrhœa, which continued, and he soon sank.	contained about a quart of curdy pus and fibrine. Colon and cœcum extensively ulcerated. Spleen friable. Kidneys rather large, with slightly adherent capsules.	
43	D. Clayton.	49	M.	Admitted December 7th, having suffered from palpitation and dyspnœa since June; never had rheumatism; no previous illness; his symptoms had come on quite gradually. A double bruit at base of heart, which was enlarged, and its action increased. Urine scanty, loaded, albuminous. Breathing soon became very laboured. He died in 1 week.	General anasarca. Left lung compressed by copious pleural exudation; a largish patch of thickening on visceral pleura. Right lung emphysematous, and some extravasation in it. Heart very large; white patch on its surface. Vascular walls somewhat thickened. Valves natural, except some slight thickening of the mitral. Commencement of aorta exceedingly thickened and indurated by calcareous and atheromatous deposit. Liver very large, congested, its edges rounded; a white patch in its capsule. Spleen congested; capsule thickened. Kidneys large and heavy; some horny deposits in their pyramids. Blood generally very fluid.	Rugæ strongly marked. Cavity quite empty. Whole surface throughout of a dark deep red, with only a little alkaline mucus in the furrows between the rugæ. Splenic region tubes healthy. Same in mid and pyloric. The capillaries were most strikingly and beautifully filled with blood, in every part, forming a fine plexus with very elongated meshes surrounding the tubes. Those at the mucous surface were much longer, and more highly congested than the vertical intertubular capillaries; there were numerous extravasations of blood around them, just beneath the basement membrane. There was no appearance of exudation taking place, the congestion, though so extreme, was in all probability passive from reflux of blood from the congested liver.
44	M. Burne.	27	F.	Is well in summer; has winter cough last four years. Gets no sleep; no appetite. Face and lips congested; breathing hurried. Signs in	Body well formed; of healthy aspect. Lungs do not collapse; very emphysematous, in some parts œdematous; lining membrane of bronchial tubes not congested; in	Splenic region—tubes extremely obscured by interstitial nuclear and fibroid formation; nuclear formations also in sub-tubular tissue; in some parts the tubes are exceedingly wasted, and their place occupied by massy

				lungs of capillary bronchitis, and of some consolidation. Dyspnœa increased, and she died in 24 hours.	some parts the lung tissue was in some degree solidified. Several fibrinous black nodules here and there, and interstitial black deposit. Mitral orifice is irregular, and its auricular surface is covered by a ring of granulations; the posterior flap is thickened, the anterior much shortened; other valves healthy. Seven large margins of lobules fatty. Kidneys hard and congested.	and extensive nuclear formations. Mid region—tubes much wasted, though still discernable; they are seen degenerating fattily amid a mass of nuclear and fibroid stuff, which is deposited here and there at the basis also of the tubes. Basement membrane absent. Pyloric region—tubes involved in abundant fibroid and nuclear formation, which has thickened the sub-tubular tissue also; tubes manifestly degenerating. Stomach contracted in middle; injected here and there; contained grayish acid chyme.
45	S. Howlett.	55	F.	Footman.—Has had good health in general. Had an attack "on lungs" 9 years ago; is subject to relaxed bowels. Skin cold; pulse rather week, quick; dyspnœa urgent; face dusky. Has spit up dark-coloured blood last few hours. Signs of consolidation in left front. He sank, and died rapidly.	A fine well made person. Upper lobe of left lung softened so as to break down readily, containing puriform matter. The other lobes of this lung and the whole of the right were highly gorged with bloody serum, and exhibited everywhere patches of dark congestion. Blood dark and fluid. Gall-bladder full of calculi. Other organs healthy.	Mucous membrane lined internally with greyish tenacious mucous. Mid region much injected with blood; black spots here and there. Splenic region—tubes in general tolerably healthy, but more or less wasted at their lower parts. Mid region—tubes healthy; surface covered by tenacious mucous containing orange pigment masses. Pyloric region covered by similar mucus; tubes extremely obscured by interstitial nuclear, granular and fibroid deposit, at least in some parts, others were found much less affected. Some separate nuclear masses encroached on the lower end of the tubes.
46	W. Barrett.	36	M.	Admitted emaciated, extremely depressed, suffering long from cough, and confined to bed with increasing weakness. Pulse quick; skin dusky; appetite good; took	Body in good condition. A large strumous abscess extending all over left elbow, not into joint. Some emphysema and a few tubercles in the upper part of right lung. Some in left. Mucous	Reaction feebly acid. Splenic region—tubes more or less obscured by nuclear and fibroid intertubular deposit; separate nuclear deposits in some parts; basement membrane of surface in several parts lost. Mid region—tubes in some parts healthy, in others

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				meat and wine. Sputa purulent and nummulated. He continued weak and low, with hectic. Sank in 10 days.	membrane of bronchi much inflamed and loaded with muco-pus. Strumous deposit in supra-renal bodies. Other organs tolerably healthy.	affected as those of splenic. Pyloric region—tubes much wasted and obscured by interstitial nuclear and fibroid deposit; some defined nuclear masses existed in the substance of the mucous membrane.
47	M. Williams.	30	M.	Admitted moribund, having suffered some time with cough and expectoration, with emaciation and increasing debility. He died in about 5 days.	Body in good condition. Tubercles and vomicæ in both lungs, with numerous pleural adhesions. Heart healthy. Numerous spots of ulceration in cœcum; tubercles in sub-mucous tissue of ileum. Liver, spleen, and kidneys healthy.	Reaction highly acid. Organ empty (as received). Mucous surface pale, mammillated in the mid-region. In all the splenic, part of the mid, and even of the pyloric region, the mucous membrane was in a very great degree destroyed by self-digestion, and the blood in the vessels changed to a dark-orange pigment. The tubes in the unaltered mucous membrane of the mid and pyloric regions were tolerably healthy.
48	Jos. Moore.	24	M.	Was ill only one week with catarrh and pain at lower part of chest. Had pyrexia and dyspnœa, with 5 crepitations at base of both lungs. Urine scanty and loaded; not albuminous. Expectoration abundant and frothy, afterwards became tinged with bright-coloured blood. He took a little antimony, afterwards calomel and opium, and was cupped; but the breathing became more embarrassed, and he died in 1 week.	Body in good condition. Both lungs much congested, at back part particularly, where there were small spots of extravasation. Heart very large; cavities dilated much; walls thickened. Valves healthy. Liver, spleen, intestines, healthy. Kidneys large, coarse, congested.	Reaction in no part acid. Mid and splenic regions highly congested, and covered with tenacious mucus containing black matter; pyloric region less congested, also covered with mucus which contained less black matter. The black matter consisted of yellow pigment. The tubes were very healthy in all the regions; some sub-tubular nuclear deposits existed in the splenic.
49	J. Marwood.	49	M.	Admitted, having had cough	Body largely made; integuments	Aspect of mucous membrane not healthy;

				and short breath 3 or 4 months, dropsy of legs for 1 week. No previous illness; no rheumatic tendency. Orthopnœa. Face pale and puffy. Urine scanty, clear, albuminous. Heart's sound remarkably weak and distant. Very little breath-sound in back, but coarse moist râles. Pericardial friction was detected after 2 days; and extended cardiac dulness. Same night he had a fit; right arm paralysed; coma increased; and he died in 5 days after admission.	anasarcous. Lungs rather emphysematous and œdematous; lower and posterior part of right partially consolidated and softened. Serous and fibrinous effusion in pericardium. Valves of heart dilated, and the anterior valve orifices enlarged. There was atheroma in mitral valve, and induration of the aortic flaps at their base, to a great amount. Peritonitis had taken place to some extent. Kidneys large, and contained one or two large cysts with firm fibrous walls; one of these was filled with a solid, fatty matter; surface of one kidney highly granular at one part. Liver large, with rounded margins and slight opacity of capsule. Spleen large, firm, and hard. There was a small empty old cyst in the right cortical stratum. Increased quantity of clear fluid in cerebral ventricles. Larger arteries at base of brain very atheromatous; aorta slightly. Blood generally very fluid.	it appears mottled and not smooth. Reaction not acid. An ulcer, the size of a half-crown piece, on posterior wall, at about the junction of the splenic and mid regions; it exposed the fibres of the muscular coat: close by it was the firm cicatrix of a former ulcer. On the anterior wall there was a smaller oblong ulcer, which had not penetrated deeper than the submucous tissue. Both ulcers had clean cut, adherent margins. Splenic region—tubes tolerably healthy; some nuclear masses at their bases, extending up among the tubes; capillaries much injected. Mid region—tubes completely atrophied, replaced by nuclear and fibroid deposit; basement membrane sometimes present, sometimes lost. Pyloric region darkened extensively by black pigment; tubes very much obscured by interstitial fibroid and nuclear deposit, and degenerating fattily. The black pigment formed masses lying in the superficial stratum of the mucous membrane.
50	C. Pearce.	11	M.	Suffered 1 year with symptoms of stone in bladder. Urine occasionally bloody, thick, muco-purulent, and alkaline. Micturition very frequent and painful. No stone could be detected by sound-	Body greatly emaciated. Peritonitis with fibrinous and purulent exudation. A large abscess in psoas iliac muscle, but not connected with diseased bone. Bladder divided into two cavities by a septum; much induration around	Mucous surface quite pale; reaction faintly acid. Splenic region—tubes tolerably healthy, but obscured by interstitial nuclear deposit. Mid-region in similar state. Pyloric also. Basement membrane in the two latter regions not distinct.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				ing. He was very pale and thin. An abscess formed and presented in right iliac region; it burst of itself; after which he sank rapidly, and died 6 weeks after admission.	its base. Mucous lining destroyed down to the muscular fibres. An ulcerated opening in the posterior part of upper vesical cavity. Kidneys in a most advanced stage of scrofulous disease. Other organs healthy.	
51	E. Stokes.	34	F.	A diminutive person, suffering 5 or 6 months from fits, in which she was quite unconscious. Amenorrhœa complete last 7 months. Was anæmic, ill-fed; had leucorrhœa and pruritus vulvæ. After 24 days she had a prolonged epileptic attack; after which she sank into coma and died.	Body somewhat emaciated Arachnoid thickened in parts, with some yellow millet seed deposits upon it; its vessels congested. There were three or four calcareous masses in the arachnoid at the posterior part of right hemisphere. At posterior part of left there was a thickened mass of arachnoid, with much injection round it, and much fluid in its meshes: with this there was a kind of cyst connected which had caused some wasting of the convolutions. Brain natural; rather injected; two or three drachms of clear fluid in ventricles. Other organs all tolerably healthy.	Stomach extremely contracted; rugæ very marked; no trace of mammillation. Surface pale; reaction not acid. Splenic region—tubes in some parts tolerably healthy, in others well nigh obliterated by diffused nuclear deposits. There are more circumscribed deposits seen occasionally either in the deeper, or more superficial part of the mucous tissue; in the latter situation they cause depressions, such as exist in the mammillated state. Mid region—tubes healthy, but obscured at their bases by fibroid formation. Pyloric region—tubes very healthy, but rather obscured by fibroid formation extending up from the sub-tubular tissue.
52	F. Harris.	2¾	M.	Had had tænia; head enlarged and square shaped, probably from some effusion into ventricles. He got an attack of double pleurisy, of which he died after some days. Was very anæmic.	Body emaciated and pale. Lungs congested, but crepitant; compressed in some measure by abundant fibrinous and purulent exudation in both pleuræ. All the viscera healthy. Brain not examined.	Mucous surface quite pale; reaction acid; cavity empty. Splenic, mid, and pyloric regions all quite healthy; tubes natural, and their epithelium abundant.

53	C. Bishop.	46	F.	Cook.—Quite well until 4 years ago, when catamenia became irregular. Had then pain in head, giddiness and sickness; she got better until 6 months ago, when she had a fit; several have occurred since. Healthy aspect, good colour. Right side weaker than left, much pain in head, urine not albuminous. Left arm became rather rigid; she had several convulsive attacks, eyesight failed, and she died in about 6 weeks after admission; 10 days before death a carbuncle formed on neck.	An enormous quantity of fat on abdomen and among the viscera. Lungs and heart healthy, except a few cretaceous deposits in the former. Liver rather granular at anterior part, elsewhere healthy. Kidneys healthy. A patch of inflammatory softening in right corpus striatum and optic thalamus: an increased quantity of fluid in ventricles.	Surface quite pale, mammillated towards pylorus; covered with black matter, consisting of epithelial débris, remains of food, yellow pigment masses, and numerous sarcinæ. Splenic region—tissue altered by self-digestion, but the tubes seem to have been healthy. Mid region—tubes in some parts healthy, in others pervaded by diffused nuclear formation; or presenting among themselves or at their bases more circumscribed nuclear deposits. Pyloric region—the tubular tissue is in nearly the same state.
54	J. Chorley.	24	M.	Carpenter. — Relatives healthy. Has generally had good health. About 4 weeks ago he began to have pain in back and right side, and soon after his abdomen began to swell. Abdomen tender and distended, appetite bad, pulse 105, some cough and mucous expectoration. An indistinct tumour was observed in the upper and left part of abdomen; it did not increase remarkably, nor was there any great amount of ascites. The febrile state continued; he	Body emaciated, finely made, anæmic. Right pleura coated with areolated false membrane, and containing fluid. Left, coated similarly to a less extent, chiefly upon the diaphragm. Right lung œdematous at back part, somewhat congested. Left lung healthy, except a few sub-pleural miliary tubercles. Heart, kidneys, liver and spleen, healthy. There was an enormous quantity of scrofulous deposit in the sub-peritoneal tissue everywhere, causing all the viscera to be matted together into one mass. The intestines were ab-	Mucous surface very dark in the splenic region, and gradually becomes less so towards the pylorus; it looks uneven, is covered near the junction of splenic and mid regions by yellowish tenacious alkaline mucus, containing prisms of triple phosphate. Splenic region—tubes much obscured and atrophied by interstitial nuclear and fibroid formation, numerous subtubular nuclear deposits exist. Mid region—tubes in much the same state as splenic; pigmentary deposits in the mucous and sub-mucous tissue. Pyloric region—tubes in great part obliterated by fibroid formation.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				became gradually weaker, and died about 2 months after admission. Signs of pleurisy had been observed on both sides. His digestion at one time was pretty good.	solutely inseparable from each other.	
55	Millen.	63	M.	A grazier in good circumstances, who suddenly dropped down dead after having travelled 80 miles in a second class carriage in very severe weather. He had had a "fit" before. Arcus senilis existed.	Body fat, much fat in sub-serous tissue of abdomen and on heart. One slight white pericardial patch. Lungs healthy, except a little cretaceous deposit at the apices and some surrounding puckering. Heart very large, its valves appeared healthy, mitral orifice rather dilated. Walls of left ventricle not thickened, but presenting numerous patches of complete fatty degeneration. Liver, kidneys, and spleen, healthy.	Reaction acid. Mucus membrane injected at various spots, slightly mammillated in pyloric region. Splenic region—tubes tolerably healthy, a notable amount of nuclear formation in the subtubular tissue. Mid region—tubes perfectly healthy. Pyloric region—tubes tolerably healthy, more or less obscured by fibroid formation, their lower ends convoluted or drawn up into groups.
56	C. D.	1	F.	Ill 5 weeks, had at first croupy cough and fever, with convulsions and opisthotonos, afterwards some retching and sickness. She did not die comatose. No food was taken for at least 24 hours before death.	Kidneys enlarged, mottled. Lungs and heart healthy. Liver gorged with bile.	Surface quite pale, cavity empty. Reaction acid. Splenic region—mucous membrane almost entirely gone, sub-mucous tissue exposed, rendered translucent, blood in its vessels converted into yellow pigment. Mid region and pyloric—tubes quite healthy.
57	L. Clarke.	47	F.	Had cough and expectoration 8 or 9 months. Bowels open. Tongue furred. No sleep. No appetite. Pulse rapid and	Body greatly emaciated. Right pleura obliterated. A cretaceous obsolete scrofulous mass, and an empty vomica in upper part of	Mucous surface pinkish from injection, covered in most part of its extent by a tenacious chocolate-coloured mucus, which consists of a viscous plasma of faint acid

				feeble. Respiration in both sides deficient. Suffering much mental distress from death of husband. In spite of wine she declined, was very low and nervous, did not sleep; appetite was quite lost, so that at last she could take nothing but wine. Died 5 weeks after admission.	right lung. In left lung at upper part there was also a vomica and consolidation. White patches and streaks on pericardium. Right ventricle dilated, and anterior valve orifice enlarged. Some atheroma in origin of aorta and in mitral flaps. Kidneys gorged with blood, cortices mottled and rather fawn coloured. Some small cysts in broad ligament of uterus. Other viscera healthy.	reaction, imbedding multitudes of nuclear corpuscles, some celloid particles, and débris of food. Splenic region—tubes tolerably healthy, epithelium in lower part fatty. Mid region—tubes utterly obscured, if not completely atrophied by interstitial nuclear deposit. Pyloric region—tubes tolerably healthy, but much obscured by nuclear and fibroid formation.
58	Thos. Sheate.	57	M.	History of pleurisy 5 weeks before admission. Cough and muco-purulent expectoration. Tongue cracked and furred, brown. Pulse 100, no sleep; skin hot and dry. Signs of consolidation appeared in lower part of right side, and in right apex. Some delirium appeared at night; he became very drowsy, breath most offensive. Died in 17 days from admission.	Body in good condition. Left lung very œdematous. Right pleural cavity obliterated, all but a largish cavity at the upper and back part, which was full of purulent fluid, and lined with rather firm, recent fibrine. Upper part of right lung puckered, and contained three or four masses of cretaceous and putty-like scrofulous deposit. Middle and lower parts of right lung very much condensed, and contained several smallish-sized abscesses, full of fetid matter; there was no tubercle in their vicinity. Mucous membrane of bronchi inflamed. Heart healthy. Kidneys firm, with a yellowish cortex. Spleen pulpy, capsule thickened. Other organs natural.	Mucous surface pale, covered with pinkish, faintly acid mucus. Some mammillation in mid region about great curvature. Splenic region—tubes very healthy, slight trace of nuclear deposits at base of tubes. Mid region—tubes very healthy, but in some measure obscured by interstitial nuclear and fibroid formation, which is also going on in the subtubular tissue. Pyloric region—tubes tolerably healthy.

	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
59	J. Greenfield.	53	M.	Systolic mitral murmur; liver enlarged; pain referred to it; effusion into both pleuræ; some dropsy. After having suffered from the effects of these conditions, he improved and was discharged; but after 14 months, dropsy came on again, and dyspnœa. Heart's action was irregular; pulse weak; veins of neck distended; urine deep coloured and clear. He died in a few days.	Gray thin hair; universal jaundice; anasarca of legs; much yellow fluid in peritonæum; heart very large; valves healthy; a white patch on surface; some opaque thickening of endocardium of left ventricle; lower lobe of left lung much compressed by abundant pleural effusion; upper pretty healthy; right lung everywhere adherent by old attachments to walls of chest, except below and behind, compressed, highly congested, upper lobe softened; capsule of liver much thickened; parenchyma dense, and yellow, and firm. Kidneys seemed tolerably healthy, but were unduly firm.	Surface in splenic and mid regions very dark, almost black in spots; it contained a large quantity of whitish chyme, highly acid. Splenic region—tubes almost utterly broken up, and reduced to a mass of nuclei and granular globules, mingled throughout with black pigment globules. Mid region—tubes in a very great measure broken up, and their place occupied by diffused nuclei and celloid substance; the basement membrane, however, was often distinct in this and the splenic region. Pyloric region—villi beautifully distinct, with their basement membrane, but tubes utterly obscured, if not obliterated, by circumscribed and diffused nuclear deposits. There was no pigment in this part.
60	W. Hopkins.	41	M.	For 3 weeks had pain and swelling in outer side of left thigh, which obliged him to leave his work as horse-keeper. Health has been generally good, but has not had substantial food last year; lives in a bad, dirty neighbourhood. Abscess was opened, but diffuse inflammation came on, which spread to the knee-joint. He sank, and died 1 month after admission.	Lungs contained numerous small secondary deposits in an early stage; liver very fatty; heart and kidneys healthy; synovial membrane of knee-joint inflamed, with purulent fibrinous exudation extending to it along the periosteum of the femur from the seat of the abscess.	Surface of dull pinky tint. It contained a dark acid fluid. Splenic region—mucous membrane rather thin; tubes have a decided tendency to atrophy, some subtubular nuclear deposits encroaching on tubes. Mid region—tubes generally healthy; they are shortened and obscured in some spots, corresponding to mammillating depressions. Pyloric region—tubes tolerably healthy, but much obscured by nuclear and fibroid formation, which involves also the corium.

61	C. Gillmore.	29	F.	Aspect tolerably healthy; not anæmic. Dysphagia 4 months; a mass of enlarged glands on left side of neck; much mucous matter rejected, in which were seen numerous large cells, opaque with oil molecules, apparently from the gastric tubes; reaction of matters vomited not acid (on one trial). Always delicate. Digestion pretty good until last 12 months; during that time very bad; beef-tea causes a burning sensation in stomach. After some days, much bloody fluid was brought up; she became less and less able to swallow, and died 6 weeks after admission, greatly debilitated.	There was an encephaloid tumour on the left side of the neck, pressing on the œsophagus, and causing ulceration of the upper third of this canal, and of the glottis, and parts around. The middle of the œsophagus, for about 2 inches, was not more than $\frac{1}{3}$ inch diameter, and opened by a slit-like orifice into the inferior part, which appears natural, but is choked up by a thick sebaceous matter, consisting of altered epithelium. Heart, kidneys, and liver healthy; right lung contained some tubercles at apex; left was full of tubercles, with a small cavity in apex.	Stomach rather large, elongated, very much depressed, contracted in the middle; contained much turbid, dirty yellow, acid fluid. Surface palish. Splenic region—tubes pretty healthy, but tending to disintegrate, with considerable subtubular nuclear deposits, encroaching sometimes on the tubes. Mid region—tubes healthy, but epithelium very fatty and wasted. Pyloric region—tubes tolerably healthy, but partly obscured by large nuclear deposits occupying the substances of the mucous membrane, as well as diffused more uniformly between and among the tubes.
62	Ann Rolf.	74	F.	Admitted in a state of insensibility; vomiting frequently. She fell down suddenly while walking across a room. Pupils contracted and insensible. Died next day.	Body emaciated; angular dorsal curvature; heart healthy; lungs pretty healthy in front, but back parts much engorged and condensed; liver healthy; both kidneys granular; a clot of blood and some serum in left ventricle of brain, and an effusion of blood under right side of cerebellum.	Surface pale, covered with much slimy, transparent mucus, highly acid. Some mammillation in mid and pyloric regions. On posterior surface of pyloric third, near its junction with the mid, there is an ulcer with smooth, thinned edges, of squarish shape, just exposing a vessel in the sub-mucous tissue. Tubes in all the three regions very healthy.
63	R. Nelson.	52	M.	In early life a jockey, latterly a nurse. Has been operated on for fistula in ano;	Tubercles and vomicæ in both lungs, with much inflammation; liver rather granular at anterior	Surface of dull, dirty pink tint, cavity nearly empty, reaction feebly acid. Splenic region—tubes more or less wasted, some-

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				last 9 weeks had had bleeding from anus; cough, expectoration, diarrhœa, and emaciation about 3 months; signs of mischief in left chest; urine not albuminous; diarrhœa came on; he sank and died in a few days after admission.	part, fatty, and pale; heart healthy; kidneys highly granular; mucous membrane of rectum affected by old inflammation of dark slaty colour; sub-mucous tissue much thickened, some spots of ulceration, and some minute abscesses.	times completely, their place occupied by nuclear and fibroid deposit. Mid region—tubes very much atrophied by diffused nuclear and fibroid formation, with large nuclear deposits both at their bases and among the tubes. Pyloric region—tubes very much, almost entirely, atrophied by interstitial fibroid formation, with large nuclear deposits in the substance of the mucous membrane.
64	M. Jones.	25	F.	Housemaid. Has generally had very good health. Ill 11 days. A mottled rash on chest; abdomen tympanitic and rather tender; urine albuminous; it continued so throughout until her death, about 10 days after admission.	Body well made. Lower lobe of left lung highly congested, softened, and containing little air; right lung in nearly same state behind; heart and liver healthy; kidneys hard, but healthy in appearance; 3 ulcerated patches of Pyer's gland in ileum; rest of canal healthy.	Surface covered with reddish non-acid fluid; cavity nearly empty. Splenic region much injected; tubes in some parts tolerably healthy, in others much obscured or even wasted by sub-tubular and intervening nuclear and fibroid formation; here and there there are nuclear masses in the midst of the tubes. Mid region—tubes very healthy, but slightly obscured by fibroid formation. Pyloric region—tubes tolerably healthy, their lower ends in some measure convoluted.
65	W. Golding.	17	M.	Recent inguinal hernia; operation; signs of peritonitis; vomiting; death. Vomited fluid strongly acid.	Body rather decomposed; traces of peritonitis; adhesions and puriform matter round intestines. Heart, liver, and kidneys healthy; both lungs very much engorged at back parts. A portion of ileum, 1 inch long, was dead and sloughy; the part above the stricture was highly congested.	Cavity contained a large quantity of dark grumous, scarcely acid fluid; in this were numerous masses of dark yellow matter. Splenic region—tubes tolerably healthy, some nuclear masses encroaching on bases of tubes. Mid region—tubes in some parts very healthy, in others invaded by fibroid formation, extending upwards from the corium. Pyloric region—tubes generally healthy; but there are some large nuclear deposits between them, and at their bases.

66	Thomas Bacon.	41	M.	Ill 8 months, with symptoms of pulmonary phthisis. Anæmic aspect. There were all the signs of large cavities in the lungs.	Body much emaciated. Liver and kidneys pale, but healthy.	Surface pale, covered with tenacious non-acid mucus. Splenic and mid regions—tubes very healthy. Pyloric region—tubes very much obscured, if not destroyed, amid an abundant infiltration of nuclei and fibroid tissue; there were large nuclear formations in the sub-tubular tissue and corium. Lower ends of tubes often gathered up into opaque bunches of convolutions. Some parts much more affected than others.
67	A. Felby.	34	F.	A servant, single. — Had suffered with symptoms of bronchitis 17 days, had similar attacks before.	Lungs highly œdematous, congested; a little old tubercle at apices, general adhesions of both pleuræ. Heart, liver, kidneys, and spleen, healthy. Anterior edge of liver rounded, somewhat; surface of capsule thickened, and adherent to diaphragm. Body rather thin and pallid.	Surface smeared with acid mucus, most abundant in pyloric region; the mucus contains numerous sarcinæ. Splenic and mid regions—tubes very healthy. Pyloric region—tubes not healthy, their lower parts convoluted, obscured by interstitial nuclear and fibroid formation.
68	J. Oates.	48	M.	Before his admission bruised his chest severely by a fall, which caused much extravasation of blood over the right ribs. He had suffered from cough and pain in that side a long time, was exceedingly weak, and had ruined his health by drinking. He looked 10 years older than he said he was. A small venesection relieved his dyspnœa much, but he soon sank into a state of prostration, in which he sunk.	Body in good condition, muscular. A large quantity of turbid, dirty greenish sero-purulent fluid in right pleural cavity, both layers coated with recent lymph. Ribs not injured. Both lungs quite healthy. Heart rather large, but healthy. All the abdominal viscera healthy.	Surface darkish, except near pylorus; marbled aspect about lesser curvature; a kidney-shaped ulcer on posterior wall near the lesser curvature, and several dark red spots in its vicinity. Reaction feebly acid. Splenic region—tubes seem to be tending to disintegrate. Mid region—tubes indistinct, but epithelium abundant; there are some circumscribed nuclear deposits, and diffuse nuclear and fibroid formation. Pyloric region in same state.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
69	— Eash.	90	F.	A nurse in Marylebone Infirmary, died apparently of old age; had some slight bronchitis.	Limbs spare. Much fat on abdomen and among viscera. Both lungs œdematous and engorged posteriorly; a mass of grayish induration in anterior edge of right. Liver healthy, some chronic thickening of its capsule. Kidneys highly granular, atrophied to one half their normal size. Uterus enlarged, retroverted, its cavity much enlarged and lined by a bloody coagulum.	Contracted except in splenic region, contained some thin, feebly acid, chocolate coloured fluid. Surface throughout of a rather dirty, slaty aspect. Splenic region—mucous membrane appears thinned, tubes excessively wasted, débris remaining here and there, with fatty contents, and some rather large cystic formations; these are all imbedded in a dense roof of fibroid tissue, which is traversed by a great number of yellowish streaks, consisting of oily molecules. Basement membrane perfect, with numerous oily vesicles below it. Mid region—basement membrane perfect, tubes converted into a coarse granular and fibroid tissue, containing celloid corpuscles and much free oil. Here and there are seen groups of convolutions, the remnants of tubes whose outlet is obliterated. Pyloric region—mammillated in some degree, tubes wasted and lower ends gathered into bunches, with much granular and oily deposit under the basement membrane. There were a few massy nuclear deposits.
70	E. Hunt.	26	F.	Married. Suckling 1 year and 10 months, is extremely emaciated. Had 1 month ago an attack of acute pain in abdomen, with fever and diarrhœa. Abdomen enlarging last 3 weeks, is tense,	Great emaciation. Much clear fluid of reddish colour in both pleuræ, compressing lungs considerably. Lungs, heart, and pericardium healthy. Peritonæum much thickened, opaque, and vascular, contained 2 quarts of yellowish	Contained a great deal of acid, semi-fluid matter, like white gooseberry jam. This consisted of large quantities of débris of food, mingled with very numerous fragments of columnar epithelial lining of fossulæ. There was some fissuring of the surface in the splenic region, but no mam-

				tender, and fluctuates. Pulse 132, feeble. Urine scanty and loaded. Had scarce any appetite. She was tapped, with some relief, but 14 days after had much diarrhœa, and then complained of violent pain in stomach after taking any food. Was tapped again, with some relief, but 1 week after she was attacked with vomiting of a very large quantity of dark bloody fluid, and died.	serum. A large ovarian tumour existed on the left side, non-adherent, it consisted of a lowly organised fibrinous product. There was a similar smaller tumour on the right. Uterus and abdominal viscera healthy.	millation; the tubes were tolerably healthy, but there was some diffused nuclear deposit, and some fibroid formation encroaching on the basis of the tubes. Mid region—tubes healthy. Pyloric region—tubes more or less atrophied, with much interstitial nucleated fibroid formation. Duodenum healthy, villi opaque, with oil drops of chyle being absorbed.
71	M. A. Locke.	35	F.	A cook, subject to winter cough, admitted December 13th, having had 3 weeks ago a violent attack of dyspnœa, tightness of chest, and pain at lower border of right side. Tongue dirty, appetite bad, very thirsty; lips purple, covered with herpes. Pulse 120, small and weak. Much cough, with copious prune-juice mucous expectoration. Physical signs of broncho-pneumonia. She improved a good deal, but had some attacks of vomiting, and once frothy stools. She sank and died unexpectedly, about 1 month after admission.	Body in tolerable condition. Right lung consolidated, excavated in middle and upper parts by cavities containing offensive, purulent, and grumous matter. No tubercles. There were several very small masses of softening yellow fibrine in the lungs, and the bronchial tubes were very full of thick mucus. The right pleura was almost wholly obliterated, the left contained Oj of serous fluid, and some fibrinous exudation. Left lung healthy, except in one part about the centre, where there existed one or two softening fibrinous masses. Heart natural, but the fibrine in the right cavities was in parts softened, pasty, and of a dull yellowish-white colour. Recent	Splenic part darkened by acid acting on blood, in some degree self-digested; some mammillation about lesser curvature. Splenic region—tubes tolerably healthy, but altered by self-digestion; some black pigment grains in their interstices. Mid region—tubes very tolerably healthy, a few small subtubular nuclear deposits. Pyloric region—tubes much obscured, amid a granulous and oily fibroid stroma, and more or less atrophied. Mucus on surface consists of tenacious plasma imbedding granular matter, altered nuclei, large cells from the tube and flakes of columnar epithelium; it is much more decidedly acid in the splenic than in the mid region. This abnormal mucus seems to be an unhealthy secretion from the tubes.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
					peritonitis, with serous and fibrinous effusion. Liver and spleen much softened. Kidneys large and glossy. Uterus and appendages natural.	
72	— Tucker.	64	M.	Cough and bronchial expectoration during the last winter; hæmoptysis occurred some time ago, and again to a greater amount shortly before death.	Heart healthy. Lungs universally adherent to walls of chest; no tubercles; some masses of induration in various parts; much œdema of pulmonary tissue. Kidneys healthy. Liver tolerably healthy; a patch of thickening on its surface. Spleen small and soft, with surrounding adhesions.	Surface generally pale and smooth; marked mammillation towards pyloric region; — this part covered with viscid, glassy, acid mucus. Splenic region—tubes pretty healthy, but altered by self-digestion, dark pigment granules are dispersed through the tissue. Mid region — tubes very tolerably healthy. Pyloric region—tubes very much obscured by interstitial fibroid formation.
73	— Wiffin.	2	M.	Had scarlatina, ulcerated sore throat, and swelled glands; he became debilitated, and kept screaming continually till near death. Urine was albuminous; but there was no marked dropsy. He took only beef-tea and milk for some days.	Lungs collapsed at some parts near the borders; rather condensed at back parts, but still crepitant; traces of bronchitis. Heart, kidneys, spleen, and liver, healthy. Brain pale and wet. Skin and all the organs very pale.	Cavity empty; reaction very feebly acid. Splenic, mid, and pyloric regions show tubes quite healthy. Some nuclear deposits in splenic region at bases of tubes.
74	— Mills.	68	M.	Was an habitual sufferer from rheumatism; got an attack of dyspnœa, with symptoms resembling those of acute bronchitis, and simultaneous disappearance of pains from the joints. He was bled	Lungs highly emphysematous, and œdematous. Bronchi somewhat inflamed. Heart's muscular tissue soft; one aortic flap stiffened by a ridge of bony deposit, the other valves healthy. Liver and spleen healthy. Kidneys very	Surface in splenic region caused by fluid of acid reaction, and darkened here and there by altered blood. Part of the mid and of the pyloric regions are quite pale, and covered by dense non-acid mucus. Splenic region—tubes seem to have been tolerably healthy, but are much altered by self-

				with relief, but afterwards got worse, sank, and died.	much wasted by large cysts formed in their substance.	digestion. Mid region — tubes generally very healthy. Pyloric region—tubes imbedded amid much nucleated fibroid tissue; their lower ends in process of being gathered into branches.
75	M. E. Crawford.	4 months.	F.	Ill 1 month, with so-called decline. Bowels relaxed; bilious fluid stools.	Body pale; very much wasted. Lungs, heart, liver, spleen, kidneys, all healthy. The lower part of ileum had its mucous lining softened, and covered with bile-tinged and slimy pale mucus; it was not materially injected. The large intestine was darker externally than natural; the mucous membrane of the cæcum was of a slaty colour, and presented numerous spots of inflammatory injection, which in the lower part were converted into actual ulcers. Whole mucous tissue thickened.	Surface quite pale, covered with tenacious non-acid mucus. Tubes healthy, in all the three regions.
76	W. Whyles.	40	M.	Had good health, except a hydrocele, which had been cured 2 or 3 years before. Without previous ailment, except pain in the back occasionally, his legs had begun to swell, and face also. Aspect sallow, with red patches on cheeks. Pulse quick, regular. Tongue pretty clean. No evidence of disease in heart or lungs. Urine not albuminous, but he had all the	Body well made. Left leg inflamed; muscles softened, and containing much semi-purulent and bloody fluid. ℥vj of bloody fluid in left pleura. Both lungs at their lower part of a very dark, dull hue, softened. Much cretaceous matter in some bronchial glands. Heart very soft and flabby; cavities somewhat dilated; right anterior ventricular orifice very large. Endocardium blood-stained. Aorta atheromatous. Liver	Surface pale throughout, covered with a thin layer of acid slightly viscid mucus, containing much columnar and tubular epithelium. Splenic region—tubes appear atrophied, wider apart than natural, with much coarse nuclear and granular matter in their interstices. Mid region — tubes in process of being atrophied; they lie buried amid a mass of nucleated fibroid matter, in which are seen numerous fat-cells; they do not form a regular continuous row. Pyloric region—tubes in same state, and their lower parts gathered up into bunches.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				aspect of a man suffering from renal disease. After about 14 days, diffuse cellular inflammation came on, urine became albuminous and bloody, he sank and died.	soft and greasy. Kidneys rather granular and flabby, mottled and vascular their cortices greatly wasted in spots. Spleen soft. Other organs natural. There was a cavity in the liver containing matter like the débris of hydatids.	
77	G. H——.	21	M.	Suffered 2 years with dysentery, and latterly had ascites.	There was universal cancerous infiltration of the sub-peritonæal tissue, with a large mass involving the rectum and bladder. The organs were healthy.	Surface pale, covered with highly tenacious neutral mucus. Splenic and mid regions—tubes perfectly healthy. Pyloric region—tubes everywhere indistinct, buried amid an infiltration of nucleated fibroid matter; their lower ends in some spots gathered up into branches.
78	R. Stratton.	47	M.	Subject to cough for 30 years, ever since he had measles; spits blood in streaks, mingled with yellow thick sputa; his legs swell; is emaciated. Face dusky, swollen; eyes prominent. Urine not albuminous. Had some diarrhœa. Pleural friction was observed in both backs. He got worse suddenly, and died 18 days after admission.	Body œdematous; some serum in peritonæum; blood generally fluid. Slight effusion in pericardium; a long thin band of adhesion over middle of right ventricle, and adhesions also about large vessels. Right ventricle thickened and enlarged. Left of natural size. Aortic valves—only 2—thickened, their edges hardened and involuted; they were not efficient. Other valves healthy. Right lung everywhere adherent; its lower lobe gorged with black blood and serum; does not collapse; is soft and friable; its bronchial	Cavity contained a quantity of reddish chocolate-looking alkaline fluid. Mucous membrane much injected in splenic region;—in the middle it was quite of a dark red, in the pyloric of a slaty blackish-gray. Splenic region — tubes tolerably healthy, but there is a decided formation of nuclear fibroid matter, extending up from the corium among the tubes. Mid region —tubes in some parts extremely wasted by circumscribed and diffused nuclear deposits; in other parts they are tolerably healthy, and there is only some nuclear fibroid formation at the bases of the tubes, and encroaching among them. The intertubular capillaries are much injected, and

					tubes thickened and narrowed; upper lobes emphysematous. Left lung emphysematous; a small tubercular mass at its apex with puckering round it; otherwise healthy. Kidneys appeared healthy. Liver healthy.	there is in many spots a deposit of black pigment grains. Pyloric region — tubes tolerably healthy, their deep ends convoluted here and there; some interstitial fibroid formation; opaque fat masses in some of the villi, and black pigment deposited here and there.
79	J. J——	45	M.	Father of a large family. Had symptoms of phthisis some years. Suffered from diabetes 2 years. Average quantity of urine 24 pints daily; sp. gr. 1035. General anasarca at time of death.	Lungs full of tubercles; very large cavity in the left.	Reaction of surface markedly acid. Mucus of surface consists of homogeneous granular plasma, entangling débris of tubular and columnar epithelium with numerous torulæ. Splenic portion—mucous membrane rather darkened, thinned, and softened; tubes seem very healthy, but rather altered by self-digestion. Mid region — tubes very tolerably healthy, with a little interstitial nuclear formation. Pyloric region—tubes indistinct from intervening granular nucleated exudation.
80	H. Spillen.	56	M.	A potman.—Admitted unconscious, with very contracted pupils and hemiplegia of right side. Bowels freely acted on. He fell down suddenly and vomited, having, it was said, eaten a hearty dinner on the day he was attacked. He is in the habit of taking 5 pints of porter, besides gin, &c., daily. Died in 2 or 3 days.	No arcus senilis. Skin generally of a yellow hue. Dura mater very adherent to bones; vessels much congested. A thin layer of blood in sub-arachnoid tissue of cerebellum. Cerebral arteries slightly atheromatous. Central parts of brain much softened; ventricles much dilated;—left full of soft dark clot, right contained a little. Left corpus striatum and optic thalamus—broken down by extravasation of blood; and also septum lucidum. Traces of	Surface pinkish, smeared over with tenacious bile-tinged neutral mucus, in which were crystals of triple phosphate. Near the pyloric region and lesser curvature there are some spots the size of a small pea, which are incipient ulcerations, infiltrated by hæmorrhagic exudation. Splenic region—tubes very healthy. Mid region—tubes perfectly healthy. Pyloric region—tubes very much obscured, and more or less atrophied amid a copious infiltration of nuclear and granular matter; there are large nuclear deposits in the deeper layers of the membrane, containing much oily

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
					an old cicatrix in outer wall of right ventricle. Lower and posterior part of lungs softened and much engorged; some extravasation in parts. Left valve of heart thickened; slight atheroma of mitral valve and of aorta. Coronary arteries atheromatous. Liver highly scirrhotic. Capsule of spleen opaque; itself firm and hard. Kidneys congested and granular; their cortices wasted: much fat around them.	matter. In some sections, the tubes are almost wholly lost, and their places occupied by large nuclear deposits and granular and fibroid matter. In the situation of one of the small ulcers the surface was seen to be sunken, the basement membrane gone, and the tubes quite lost, while some remaining fibroid tissue was infiltrated with yellow pigment.
81	R. Randall.	17	M.	Sight failing since 6 or 7 years of age; has had some fits, and is half idiotic, otherwise has had good health. Cataract broken up. Some inflammation of eye followed, but soon yielded. 14 days after had febrile symptoms, followed by loss of power in his limbs, dilated pupils, staring eye. He rapidly got lower, speech failed and power of swallowing, and he died in 3 days.	Head well shaped. Superficial cerebral veins congested. Brain healthy. No fluid in ventricles. Lungs emphysematous and œdematous, and collapsed in some parts. Heart, liver, and kidneys healthy. Intestines, especially transverse colon, distended by flatus; Peyerian patches of ileum here and there much congested, and some large patches of inflammatory congestion in mucous membrane of cæcum and colon. Solitary glands very distinct; no congestion round them.	Cavity distended, containing grayish chyme feebly acid; surface pinkish. In splenic and mid regions—tubes tolerably healthy, but the mucous membrane is thinned at some parts, and the tubes shortened;—this change coincided in one instance with a diffused nuclear deposit in the substance of the mucous membrane. Pyloric region—lower halves of tubes so thrown into branches, that they look like conglomerate glands; they are filled with fatty epithelium. In some parts the atrophy of the tubes is more marked; there are distinct interspaces equal to two or three tubes in width, which are filled up by nuclear and fibroid formation, and there is much of the same also between the tubes.

82	J. Platt.	38	M.	Omnibus driver.—Not of steady habits; not a drunkard, but took spirits daily. Had a somewhat similar attack 16 years ago; health very good since, except slight winter catarrh. Voice very weak; much debilitated. Ill 3 weeks, having relapsed after a previous similar attack. Dyspnœa urgent; no ease in any position; legs highly œdematous. Expectoration scanty and dark. Urine loaded; not albuminous. Signs of engorgement of lungs. He got weaker, and sank in 5 days after admission.	Body pale; very robust and muscular; less anasarcous. Copious serous effusion in right, some in left pleura. Right lung consolidated to a great extent with solid exudation matter; tissue œdematous, softened, easily broken down. Left lung similarly affected, but to a less degree. Upper parts of both most affected. Heart very large; two aortic valves shrunk, thickened, and partially detached. Mitral healthy. Spleen much softened. Kidneys and liver healthy.	Cavity contained much watery and mucous fluid, slightly acid. Splenic and mid regions — tubes healthy. Pyloric region—tubes tolerably healthy, but showing a tendency to atrophy and to become bunched at their lower ends. In some parts they were more decidedly wasted. There were some small sub-tubular nuclear deposits, and more or less of diffused interstitial nuclear and fibroid formation. Duodenum injected; villi wasted and ragged.
83	M. Williams.	70	F.	Bed-ridden some months; had feverish and pulmonary symptoms; improved in a few days, so as to be able to take food well, but soon after rapidly sank. She took ℥iv of brandy 2 or 3 days before death.	Body rather wasted and pale. Lungs emphysematous and œdematous. Heart healthy, except some thinning of walls of right valve, and a little thickening of mitral flaps. Kidneys granular; rather wasted. Liver healthy-looking, but flattened with rather a large sub-serous patch of thickening. Spleen healthy and uterus.	Cavity so contracted, except at splenic end, as to resemble an intestine. Surface pale, with a little dotted injection; contains some darkish, non-acid fluid. Tubes in all three regions tolerably healthy. Some interstitial nuclear and fibroid formation in pyloric. Duodenum healthy.
84	J. Carroll.	58	M.	Had epileptoid fits 2 months, and perhaps longer, in which he had no convulsions, but was simply unconscious; there	Substance of brain healthy, but pale; ventricles distended. Lungs highly œdematous and congested at back parts. Heart strongly	Surface pale; reaction non-acid. Splenic region—tubes healthy, but their epithelium in lower ⅔ths is remarkably opaque and fatty; the fatty opacity ceases when the

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				was gradually increasing imbecility. He had hæmorrhoids, which bled much.	contracted; walls of left ventricle thick. Kidneys much wasted and highly granular. Liver and spleen healthy. Body thin and rather pale.	epithelium is diffused in the water around. Mid-region—tubes very healthy. Pyloric region—tubes gathering into bunches at some parts in their lower ends; no material increase of fibroid tissue. Duodenum healthy. There were some crystals of triple phosphate on the surface of the stomach and duodenum.
85	M. A. Harris.	10 months.	F.	A backward child; no teeth; ailing from birth; was suckled for 9 months. She had diarrhœa, and vomiting, and general emaciation.	Large intestine presented in its upper part numerous spots of tumefaction and injection, and in its lower numerous minute pinhole ulcerations. The mesenteric glands were considerably enlarged, not by scrofulous deposit. The ulcerations on the mucous membrane of the intestine showed under the microscope a distinct loss of tissue, with a large solitary gland lying in the sub-mucous tissue beneath.	Surface pale; reaction acid. Splenic region—tubes very healthy, but scarcely fully developed. Mid region—tubes quite healthy. Pyloric region—tubes quite healthy, forming a continuous row of simple parallel follicles.
86	J. Frost.	22	M.	Was taking mercury for syphilis before admission. Symptoms of fever. No sleep. Pain in head. Pulse soft, not frequent. Pupils afterwards became dilated. Tongue black, picked bed-clothes. He sank and died in 9 days after admission.	Body in good condition. Membrane of brain healthy; sinuses and cerebral veins much congested, as well as the substance of the brain. Ventricles filled with a slightly opaque serous fluid. Both lungs and heart healthy; 2 oz. of turbid fluid in pericardium; spleen soft and pulpy. Other organs healthy.	Empty, surface pale, smeared over with acid chocolate fluid, containing abundant débris of tubular epithelium, and a little pigment. Splenic and mid regions—tubes very healthy. Pyloric region—tubes not wasted; some local sub-tubular nuclear deposits, with interstitial nuclear and fibroid formation. Epithelium in lower parts very fatty.

87	W. Trowsdale.	45	M.	He had 2 or 3 attacks of cerebral symptoms, afterwards short breath, cough, and tremors; was very nervous. Had palpitation and pain in head. Appetite pretty good, took meat. He improved under treatment, but got again worse, had hallucinations before death.	Both lungs most highly emphysematous, bronchial tubes inflamed. Heart, liver, and spleen, healthy; liver much congested. Kidneys of natural size, capsules very adherent, surface uneven, some cortical tubes appear wasted, others are stuffed with dense granular matter.	Contained acid chyme. Surface in pyloric half much injected. Splenic region—tubes tolerably healthy, but much bulged and distended at their lower parts by epithelium; free fatty matter deposited underneath the basement membrane of the surface. In one part the lower ends of the tubes appeared to be giving origin to cysts. Mid region—the tubes show a tendency to waste, and to cyst formation, by distension of the blind ends; there are sub-tubular nuclear deposits, and some more diffused nuclear formation in the substance of the mucous membrane. Pyloric region—tubes fatty, and forming bunches at their lower ends, with much interstitial nuclear and fibroid formation.
88	M. A. Goose.	30	F.	Had rheumatic attack Oct. 3 and 19, after last was treated for heart disease. Ill 6 weeks, has had œdema of legs 3 weeks ago. Dry cough, dyspnœa, except when quite still. Loud mitral murmur at base and apex. Pulse 132. Enceinte 3 months. Urine high coloured, doubtfully albuminous. Much emaciated. Appearance became very bad, with heaviness and pain at epigastrium: 3 weeks after was much the same, could not take her food, dreaded to go to sleep for fear of being choked. Soon after	Body in tolerable good condition, extremities œdematous. Left pleura three parts full of fluid, much also in right. Lungs congested, œdematous. In upper part of left lung were 3 or 4 accumulations of softening fibrine and purulent fluid, surrounded by consolidated lung. No scrofulous deposit in any part. Pericardium universally adherent. Heart large, flaccid, mitral flaps rigid and opaque; bead-like fibrinous deposits along margins of aortic valves, and on auricular aspect of mitral. Kidneys, spleen, liver, healthy. Uterus equal a large orange in size,	Cavity large, containing an abundance of deeply bile-tinged acid (highly) chyme. Splenic and mid regions—rather altered by self-digestion, tubes very tolerably healthy. Pyloric region—tubes show a tendency to waste; there are nuclear formations in sub-tubular tissue encroaching among the tubes, and in some parts a good deal of interstitial fibroid formation.

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				she miscarried. Urine became scanty and highly albuminous. She soon sank.	walls thin, very firm, its lining membrane dark, and roughened by fibrinous deposit. Veins in broad ligaments and ovaries contained fibrine and puriform dark coloured fluid. Left renal vein contained fibrine undergoing change.	
89	D. Brooker.	50	M.	Of aged cachectic appearance. Admitted January 13th for difficulty in passing urine, which had increased during the preceding 14 days almost to retention. No stricture. Urine highly offensive, containing pus. On 15th, an abscess was opened in the perinæum, and much pus was let out; after 2 days urine passed by this channel, and much pus by the urethra. Died by asthenia on 24th.	Body thin. All the organs of chest and abdomen tolerably healthy. On left side of bladder a pea-sized orifice, opening into a very large abscess on left side and base of bladder, and extending into prostate. This abscess had been opened from the perinæum, the areolar tissue of which was sloughy. Urethra quite healthy.	Cavity empty, reaction neutral. Splenic and mid regions—tubes healthy. Pyloric region—tubes fatty in their lower parts, which are more or less gathered into bunches, with some interstitial nuclear and fibroid formation.
90	E. Lonergan.	24	M.	Was quite well until 10 months ago, when cough, emaciation, and debility came on. Has some anasarca, thick curdy expectoration, appetite bad. Urine highly albuminous. Appetite failed completely; he got lower, and died 1 month after admission.	Lungs riddled with vomicæ at their upper part, and full of tubercles. Kidneys enlarged and mottled.	Surface pale, healthy. Splenic region—tubes very greatly atrophied; the tissue infiltrated by nuclear deposit, both diffused and in masses. Mid region, in about the same state. Pyloric region—tubes in general very much atrophied, with considerable granular and nuclear interstitial deposit.

91	E. Taddenham.	27	F.	Ill 12 months. Breath very short, aphonia last week; has had some diarrhœa. Died in 8 days.	Body emaciated, chest very contracted. Tubercles and vomicæ in both lungs. Cordæ vocales ulcerated. Stomach and other viscera much pushed down, but apparently healthy.	Surface stained by bile, reaction feebly acid. No injection. Splenic region—tubes in some degree wasted, epithelium stunted and scanty, some interstitial fibroid formation. Mid region—tubes in some parts very much atrophied, in others less; some subtubular nuclear deposits, some interstitial fibroid formation. Pyloric region—tubes almost entirely atrophied, in some parts, amid an overwhelming infiltration of nucleated fibroid tissue, with nuclear masses in some spots among the tubes.
92	Ann James.	70	F.	Confined to bed for 4 or 5 years, had suffered long from chronic rheumatism, and had attacks of mental aberration. No dyspeptic symptoms. Died with bronchitis.	Brain wet, but healthy. Subarachnoid fluid increased. Slight thickening of arachnoid on convex surfaces of hemispheres, scarce any gland: Pacchioni. Lungs rather emphysematous, much congested and condensed at back part. Heart healthy; a white pericardial patch. Liver fatty. Spleen small, healthy. Kidneys wasted and granular. Body pale and thin, fingers and toes much distorted.	Contracted very much, contained some chocolate coloured, feebly acid fluid, fine dotted injection. Splenic region—tubes have perished extensively amid an infiltration of nuclei and granular matter, some large masses of nuclei are seen. Mid region, in nearly same state; large nuclear masses at bases of tubes. Pyloric region—tubes much obscured and wasted from the same interstitial deposit. Duodenum appeared healthy.
93	B. Patrick.	49	M.	Coachman, has lived freely. Has had an eruption and swelling of legs 3 days. Pulse full and hard. After venæsectio ad ℥xij, purpura was less, but hæmaturia came on. Urine very scanty. After cupping on the loins, blue pill and diuretics; the quantity of	Body well made, purple spots on various parts of surface. Blood extravasated at back of scalp. Brain very wet, rather more fluid than natural in ventricles, not otherwise diseased. Lungs very congested, œdematous, some extravasation in lower part of right. Heart's cavities rather dilated,	Surface much injected, rather marbled, with two patches of black staining near middle of great curvature; pyloric region covered with tenacious mucus, reaction nowhere acid. Splenic region—tubes in great measure broken up amid an infiltrating fibroid stroma, with large nuclear masses in the deeper parts of mucous membrane. Among the débris there was an oval cyst

No.	Name.	Age.	Sex.	History.—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				urine increased, but continued to contain blood. Blood has also passed in the stools and vomited. He sank and died in 15 days after admission.	otherwise normal. White pericardial patch. Liver very firm and cirrhosed, with opaque capsule. Spleen very soft, capsule opaque. Kidneys very large, smooth, with mottled surfaces and swollen cortex. Blood very fluid.	filled with granular cells. Basement membrane well marked. Mid region—tubes extremely wasted amid an infiltration of nuclei and fibroid matter, with sub-tubular nuclear deposits. Black pigment is deposited in the stained spots, chiefly between, but partly also within the tubes. Pyloric region, tissue in same state, groups of yellow pigment molecules here and there.
94	M. Ensor.	52	F.	Subject to bronchitis 3 years or more, had no dyspeptic symptoms. Sank gradually.	Body emaciated, pale. Right lung condensed in parts, and œdematous; the bronchi somewhat dilated, and their mucous lining thickened. Left lung much condensed and congested, bronchi dilated and inflamed. Heart healthy and liver. Kidneys rather wasted and granular. Spleen solid, of dirty gray aspect on anterior surface.	Splenic and mid region—tubes fairly healthy. Pyloric region—tubes more or less obscured and atrophied by interstitial nuclear deposit. Reaction acid. Duodenum healthy.
95	Ann Coles.	69	F.	Tenant of an almshouse, was 1 week in union; not ill, but feeble, and complained a little of short breath. She made no complaint of dyspepsia. Died quietly in bed.	Heart—walls flaccid, cavities loaded with blood. Lungs completely adherent to walls of chest, their upper parts full of miliary tubercles, and much condensed and consolidated. Kidneys, liver, and spleen, tolerably healthy.	Surface presents several black-stained spots, and contained much chocolate fluid of acid reaction, generally pale. Splenic region—tubes tolerably healthy, but rather altered by self-digestion; blood turned into yellow pigment. Mid region—tubes healthy, altered in some parts by self-digestion; at one dark-stained spot there were a vast number of large black grains within the tubes. Pyloric region—tubes generally healthy. Duodenum healthy.

96	W. Burnell.	16	M.	14 days ago inflammation of right leg came on without any cause. An abscess occurred in middle finger of left hand 1 week before. After admission several incisions made, much purulent discharge. Urine found albuminous. His respiration became quick, and cough came on. Pulse became excessively rapid; he got lower and died.	Body rather thin. Heart healthy, some turbid serum and films of fibrine in pericardium. Left lung much congested at back and lateral part, fibrinous layers on pleura. Right lung rather congested at back part, healthy; a cyst about the size of a walnut impacted in the hilus, and near it several enlarged glands. Kidneys, liver, and spleen, quite healthy. Pus in right sub-clavicular articulation and in right knee.	Surface smeared with a little yellow, feebly acid fluid. Tubes healthy in all the three regions, rather altered by self-digestion in a part of the splenic, which was red stained. Duodenum appears healthy to the eye, but the villi are utterly ragged and wasted, they have no basement membrane, and are full of fatty granules; reaction of the surface pretty strongly acid.
97	W. Newstead.	44	M.	A retired grocer, of full habit, subject to piles. While warm with walking was exposed to cold and damp. Afterwards had numbness of feet and legs. Has now pain in lower part of back, has lost use of legs 3 months. Urine very acid. Pulse quiet. After 14 days erysipelas attacked the left forearm, he became exhausted and sank.	Body very obese. Diffuse cellular inflammation of left arm. Brain healthy, except an increase of sub-arachnoid and ventricular fluid. Spinal meningeal vessels congested. Spinal cord healthy, except for space of 1 inch opposite 4th and 5th dorsal vertebræ, where it was much softened, but not discoloured. Spleen very soft. Kidneys soft and greasy. Bladder and other organs natural. Lungs greatly engorged. Heart flabby and soft, its lining membrane blood-stained.	Surface slightly pinkish, covered with a dirty chocolate coloured layer, consisting of columnar and tubular epithelium, remains of ingesta, and numerous dark orange pigment masses in mucous plasma. This is highly acid. Splenic region—tubes quite healthy, and little affected, though the sub-mucous tissue and fat-cells are decidedly altered by the acid. Mid region—tubes quite healthy. Pyloric region—tubes healthy, with some fibroid nucleated formation encroaching on their bases. The smell of this stomach was most peculiarly rancid and penetrating.
98	H. Knight.	62	M.	Labourer. Ailing last 3 months, but for last 18 has had incontinence of urine when the bladder has been at all full. Much emaciated lately, appetite very good. Very weak. Some diarrhœa	Body in good condition. Lungs and heart healthy. Pleuræ adherent. Peritonitis and purulent exudation in abdomen. Intestines, liver, and spleen, healthy. A circumscribed abscess at fundus of bladder; it was hypertrophied and	Surface injected in splenic region, pale elsewhere, covered with a thin layer of neutral mucus. Splenic and mid regions—tubes healthy. Pyloric region—tubes very much obscured, and in great measure atrophied by interstitial nuclear formation, with large nuclear deposits extending some dis-

No	Name.	Age.	Sex.	History—Disease fatal.	Post-mortem Examination.	Condition of Stomach.
				last few days. Has had a cough for years. Urine light coloured, soon becoming alkaline, contains pus and casts (?). The catheter drew off a good deal of urine after he had passed water. After some days, pain in the abdomen, and green vomiting came on, and he sank.	fasciculated, contained a large quantity of purulent urine, ureters and pelvis of kidneys dilated. Kidneys softened, injected with numerous small abscesses disseminated through their substance. A firm tumour of the size of a small walnut projected into the bladder; it was of prostate gland structure, contained in a firm, fibrous capsule.	tance along the bases of the tubes and among them. In one section there was a largish cyst, with a caudate offset.
09	M. Trivett.	77	F.	An inmate of the workhouse some years, had no particular symptoms of dyspepsia. Ill about 1 week, with febrile symptoms; improved, but one night was found speechless, after getting out of bed, and died in an hour.	Body rather thin and pale. Heart appeared healthy. Lungs emphysematous and œdematous. Liver and spleen tolerably healthy; kidneys also, but shrunken at one part of surface. A very large fibrous tumour in the pelvis.	Cavity much contracted in the middle; surface rather injected, especially at the pyloric part, where it is covered with some tenacious whitish mucus. No acid reaction. Splenic region—some trace of mammillation; tubes tolerably healthy, but in some spots there is an extensive infiltration of nuclear particles among them. Mid region—tubes tolerably healthy. Pyloric region—tubes much obscured by nuclear and fibroid interstitial formation, their epithelium wasted and fatty.
100	W. Smith.	13	M.	Suffered with symptoms of morbus cordæ; scrofulous 6 months or more. Appetite generally good, took food well. After the opening of a large abscess he got emaciated and weakened; feverishness came on shortly before death, and there was at last some bleeding from the wound.	Lungs congested, but free from tubercle. Heart, liver, kidneys, and spleen, healthy. Numerous enlarged glands, solid in the lower part of ileum, with much surrounding congestion. Hip-joint disorganised, cartilages destroyed, bone carious	Surface quite pale everywhere. Cavity contained a good quantity of dark acid fluid. Splenic and mid regions—tubes tolerably healthy, some abnormal fibroid and nuclear formation at their bases in some spots. Pyloric region—tubes quite healthy.

EXPLANATION OF THE PLATES

Illustrating Dr. Handfield Jones's Paper on Morbid Changes in the Mucous Membrane of the Stomach.

Fig.

1. Stomach tube, containing black pigment grains at its lower part.
2. Vertical section of splenic region of mucous membrane of the stomach, the tubes all broken up, and their débris mingled with very numerous black pigment globules. Some of the altered tissue is shewn more highly magnified at (*a*). The mucous membrane was of a very dark colour, in some spots black.
3. Vertical section of mucous membrane of stomach in mid-region. A deposit of nuclear particles is seen encroaching on the tubes.
4. Vertical section of mucous membrane in the mid-region, showing complete wasting of the tubes, and their place occupied by granular and oily detritus and fat-vesicles. The basement membrane still persists.
5. Vertical section of mucous membrane in pyloric region, the tubes much obscured and atrophied by interstitial nuclear deposit. A cystic cavity with a caudate offset is seen in the substance of the mucous membrane.
6. Vertical section of mid-region of mucous membrane of the stomach, showing the tubes utterly wasted, and replaced by fibroid tissue. At (*a*) are shown two cyst-like remnants of the tubes which were brought into view by acetic acid. The basement membrane of the surface still exists.
7. Atrophied epithelium from stomach tubes.
8. Catarrhal mucous from surface, it contains some cells from the tubes, numerous nuclei, and a columnar particle.
9. Healthy epithelium; cells from the tubes and columnar particles.
10. Vertical section of gastric mucous membrane in mid-region, showing several papilloid masses of epithelium exuding from the follicles.
11. Vertical section of upper part of mucous membrane in the mid-region, showing a cyst lying in a nuclear deposit. Diameter of cyst $\frac{1}{68}$ inch. It contains nuclei, and a clear fluid.
12. Vertical section of mucous membrane in pyloric region: the tubes in the upper part have disappeared, in the lower they are undergoing fatty degeneration. Much oily matter is dispersed through the tissue. The basement membrane is gone.
13. Vertical section of mucous membrane of mid-region of stomach. The tubes are almost entirely obliterated, and the basement membrane is lost.

EXPLANATION OF PLATES.

Fig.

14. Vertical section of mucous membrane of stomach in the mid-region. (*a*) Basement membrane. (*b*) Tubes degenerating. (*c*) Corium thickened. (*d*) Submucous tissue.
15. Remnants of three tubes breaking up into granular tracts of nuclei.
16. Vertical section of mucous membrane of stomach about the mid-region. The tissue is pervaded by nuclear deposit, and the tubes are indiscernible. Nuclei are seen also in the corium and submucous tissue. At the lower part are two opaque fatty masses; the basement membrane is seen in the upper border.
17. Vertical section showing the mucous membrane fissured in two places down to the corium.
18. Vertical section passing through a notch on surface of mucous membrane: the notched part is covered by a layer of nuclei. Tubes partially disintegrated.
19. Mucous membrane of stomach; the tubes atrophied, the whole tissue pervaded by nuclear deposit.
20. Vertical section of pyloric region, showing the villi and the nucleated substance within them. This substance was abnormally developed in the deeper part of the mucous membrane.
21. Vertical section of mucous membrane of stomach, containing a nuclear mass in its substance. The mass is in part displaced, and an empty cavity left. The surface is covered by a layer of disintegrated epithelium. (*a*) Separate nuclear particles.
22. Vertical section of mucous membrane, showing a large cystic cavity occupying its whole thickness. (*a*) Basement membrane of surface. (*b*) Mucous membrane pervaded by nuclear deposit. (*c*) Corium.

Fig. 1.

Fig. 2.

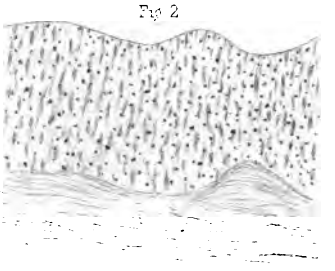


Fig. 3.

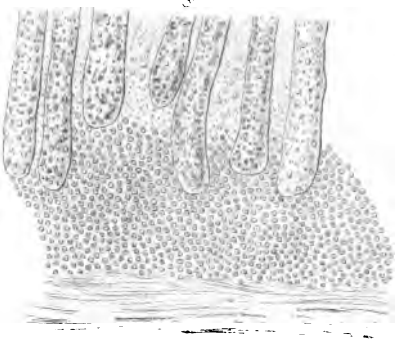


Fig. 4.

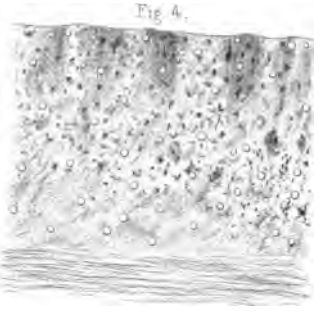
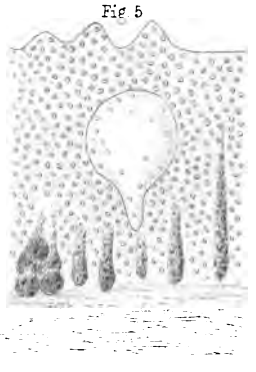
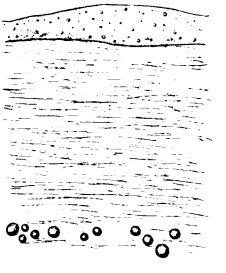


Fig. 5.





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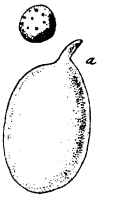
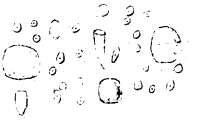


Fig. 7.

Fig. 8.



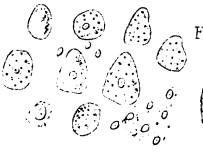


Fig. 9.

Fig. 10.

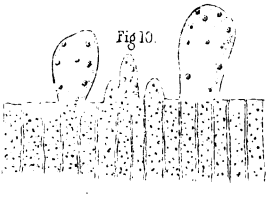
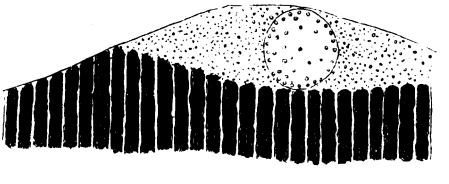
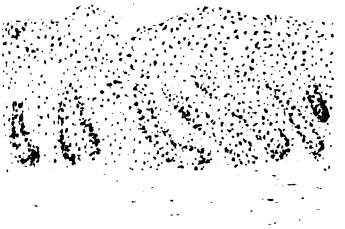
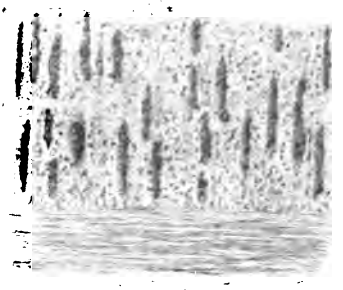
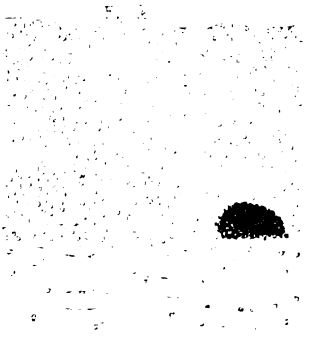


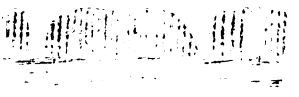
Fig. 11.

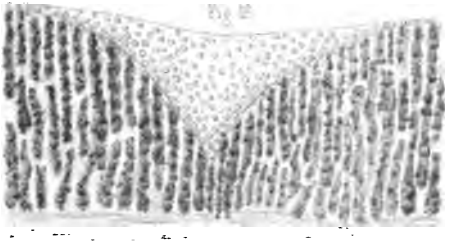


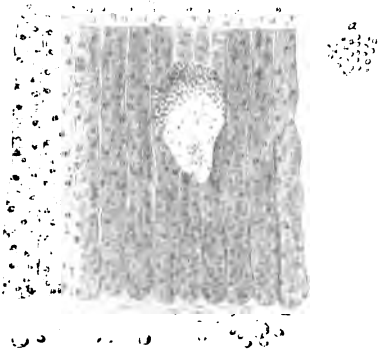


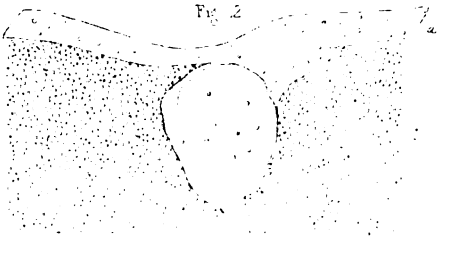












A CASE

OF

FATAL ASPHYXIA,

CAUSED BY

THE DETACHMENT OF A DISEASED BRONCHIAL GLAND WHICH WAS IMPACTED IN THE LARYNX.

BY

GEORGE EDWARDES, OF WOLVERHAMPTON,

FELLOW OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND, AND OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY OF LONDON;
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Received April 26th.—Read May 9th, 1854.

JOSEPH PERRY, about 8 years of age, of fair complexion and light hair, considered healthy but delicate looking, was playing with boys of his own age, when it is said he was offended or struck by one of his play-fellows. He ran off to tell his mother, and just as he got to his parent's house, a distance of thirty yards, he rushed towards a female, whom he met, and who thought that the child was in a fit. She carried him quickly up stairs to his mother, who charged the child with having swallowed something. This he denied in a voice sufficiently strong and distinct to be understood. His struggles became so violent that he could scarcely be held by ordinary force. The head was often thrown forcibly back, and the arms were extended occasionally by a similar strong effort. The face was discoloured, the countenance was extremely anxious, and he uttered the expression more than once, "*Mother, I shall die.*" During the struggling also, he cried out that he wanted to make water, and almost instantaneously he voided both urine and fæces. There was no cough, and the bystanders said there

was not any noise in the throat, but there were tumultuous sounds about the upper part of the belly. My partner, Dr. Bell, accompanied by Mr. Gatis, saw him about half an hour after the seizure; at that period no sound could be discovered by a hurried examination either in the trachæa or chest, except the weak action of the heart; the countenance was dark, and the child was making some feeble struggles, evidently death-throes. In the hurry of the moment there was no opportunity of getting any distinct knowledge of the previous history, beyond the surmise that the child had swallowed something.

Dr. Bell and Mr. Gatis immediately opened the trachæa, when a little air issued from the opening, artificial respiration was attempted for some time by means of a female catheter, but without effect, as the child only gave two gasps after the operation.

I. assisted in the examination of the body seventy-two hours after death. There were no marks of external violence. The jugular veins on both sides were greatly distended. On opening the chest, the lungs and heart occupied their usual position, and presented their natural appearances, except that the lungs were uniformly dark, and greatly congested. Both sides of the heart were empty, and there was an ounce of bloody serum in the pericardium. Touching the under surface of the epiglottis, and extending through the rima glottidis into the larynx, was a body about an inch in length, of irregular thickness, and presenting in its form something of an hour-glass contraction; one end was thicker and longer than the other; the substance was whitish, and covered with mucus, and in appearance much resembled a piece of chewed newspaper, being marked with black or blueish grey lines, and clots, exactly as a piece of chewed printed paper would appear. It was easily removed from its position. On a further examination it was evident that the body was a bronchial gland, broken up irregularly, and adhering at the narrow part by cellular tissue. Slitting open and tracing the trachæa, the spot from which the gland had issued, was soon found; it was

on the posterior part of the right side, just above the bronchial bifurcation. The opening was ragged and irregular, and communicated with a cavity behind, sufficiently large to contain a good sized nutmeg. The other bronchial glands were normal, the trachæa contained some frothy mucus, and the lining membrane was somewhat congested.

There appeared to be no glandular disease in any part of the body, nor was there the slightest appearance of tubercles in the lungs. The stomach contained no food, but was much distended with air.

The case is interesting, not only from its novelty, but in many points of view. Did suppuration commence within the gland, or was this a case of ulceration around the gland, detaching it from its bed and opening a passage for it into the trachæa? Again, did the gland at once pass into the trachæa, or was it a gradual process? The shape of the gland seems to indicate the latter process, and probably the blow the boy complained of, or a sudden effort at play, was the means of entirely disengaging it from the opening; the expulsive efforts afterwards forced it into the glottis and destroyed the child. In either case there appears to have been no symptoms to point out the mischief which had been going on in the child, who was represented by his parents as having been free from cough, hoarseness, or difficulty of breathing. As nearly as can be calculated, the time which elapsed between the commencement of the suffocating feeling and the death of the child, was half an hour; hence there could not have been complete closure of the glottis at first. Indeed it is a question whether the obstruction to the breathing was ever complete continuously, or was only to such an extent as to prevent that ingress and egress of air, which is necessary for the sustenance of life, each respiratory act being so imperfect that the blood by degrees became poisonous and unfitted to supply the nervous stimulus required to maintain the heart's action to transmit the blood through the lungs. Hence their dark and engorged condition after death; and hence, probably, the want of

success in the attempt to inflate the lungs after tracheotomy.

This case is also interesting in a medico-legal point of view. False accusation might easily have been made; indeed the boy in this very case complained of being ill-used by his playmates, and had any of them been much older and stronger than himself, there would have been great difficulty in convincing the parents that the death of their child had not resulted from violent and improper treatment. Suppose two persons to have been quarelling and blows were exchanged, during which a diseased bronchial gland became lodged in the glottis of one of them; he falls and struggles as if suffocating, and half an hour afterwards expires; the presumptive evidence certainly would be that the blows had killed him, and it might require a very careful *post-mortem* examination on the part of a medical witness, who had not seen the person before death, to convince himself and satisfy a jury that a little whitish elongated glandular body lying loosely in the larynx had been the cause of death.

With regard to the treatment of the present case, I think it is obvious that any means adopted at the period when Dr. Bell and Mr. Gatis saw the patient would have proved ineffectual; tracheotomy, however, appeared to offer the best chance, and probably if performed earlier, might have saved the boy's life.

The Bronchial Gland as it appeared after it was taken from the glottis.

REMARKS ON A PECULIAR FORM

OF

TUMOUR OF THE SKIN,

DENOMINATED

"PACHYDERMATOCELE,"

ILLUSTRATED BY CASES.

BY

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Received May 9th.—Read June 13th, 1854.

THE dermoid tissue is liable to a greater variety of diseases than any other in the body, and although a great number have been carefully and accurately described, there are some which have not yet received the attention of professional men. Among the latter, there appears to me to be none more remarkable than that which is the subject of the following paper.

The following five cases illustrate this peculiar state of the dermoid and subjacent tissues, and I propose to give to it the name *pachydermatocele;* all have been congenital, the disease beginning in a brown spot or mole, as such appearances are generally called when small, and increasing with the years of the individual, until, as in three of my cases, they presented hideous and disgusting deformities.

The morbid structures have all been more brown than the surrounding integuments, with a flabby feel, very like a relaxed and very emaciated mamma. In several of the cases there were two and three layers or stories, as in the one upon the neck and shoulder, resembling the fanciful and successive turns of a

tippet, or three separate folds of a rich maroon velvet curtain as may be seen in Plate II.

They do not appear to possess any great degree of vascularity, neither having veins visible upon the external surface, nor diminishing in size much after being detached from the body, therein differing essentially from the nævus maternus, or aneurism by anastomosis. The cases of the two boys, in which the disease involved extensively the scalp, one side of the face, and extended below the base of the jaw, presented by far the greatest amount of blood-vessels.

On cutting a slice transversely, or making an incision into these growths, the sub-dermoid structure to the eye seems to be hypertrophied areolar tissue, with very little evidence of blood-vessels running through it. From their general appearance and duration, there is no evidence whatever of anything malignant in their structure or tendency. In one of the cases there was a return of the same kind of tumour upon the same spot, namely the head, though to a less extent, demanding a second excision, and it returned again, and is now of the same shape and character as at first. In another patient, a boy, the same disposition to return was observed during the granulating process, but it was completely conquered by the patient and skilful application of compressed sponge and the roller bandage. In the other three cases there was no disposition whatever to a reproduction of the disease.

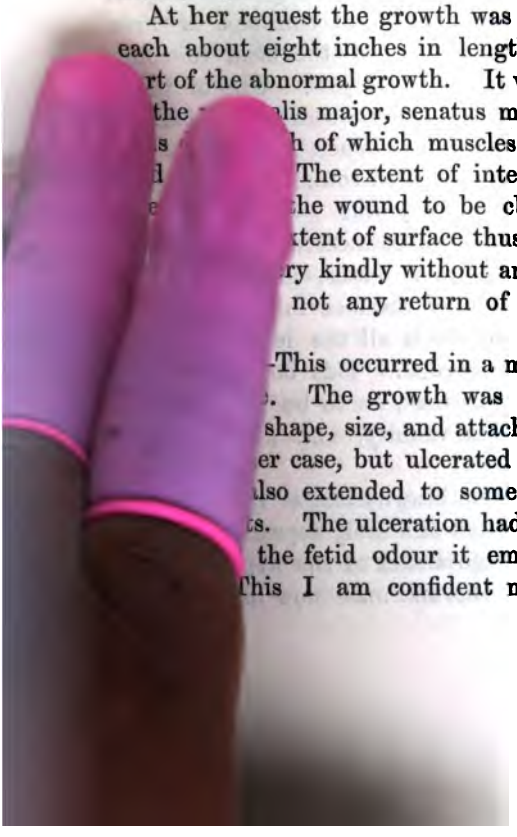
The sense of feeling was somewhat diminished below the natural standard in all of the cases. In only one was there any ulcerative action, and this arose entirely from want of attention to personal cleanliness. The largest required to be carefully washed every day with soap and water, then well dried and powdered with some farinaceous substance. If this was neglected for one day an acrid fetid discharge took place, soon leading to excoriation.

Case 1.—A young woman, æt. 24, of an excellent constitution, and uniformly good health, requested me to examine something which had existed from her earliest

recollection, and was steadily increasing in size. She said it was a swelling on the left side, not attended with the least pain, and only annoying to her from interfering with the comfortable and symmetrical arrangement of her dress. She evinced a delicacy in showing it, arising more particularly from some story of a fancy of her mother as to the cause.

On exposing it to me I found a flat tumour from four to six inches in length, and nearly the same in breadth, hanging completely pendulous. It was situated on the left side a little obliquely, about four inches below the mamma, and on a line with the axilla. It was about an inch in thickness, of a brown colour, and on closely inspecting the surface it was found to be beautifully striated, the striæ running in a serpentine manner. This was visible on both sides, and scores of dark points were distinctly to be observed sprinkled over the striæ. These spots were of the size of common and large pins' heads, and when picked out might be said to resemble dry melanotic formations. (*Vid.* Plate II.)

At her request the growth was removed by two incisions, each about eight inches in length, so as to include every rt of the abnormal growth. It was dissected from portions the lis major, senatus magnus anticus, and latissi- s h of which muscles appeared perfectly sound d The extent of integument left did not allow e he wound to be closely approximated. The tent of surface thus left uncovered granulated ry kindly without any untoward circumstance. not any return of the morbid growth.



-This occurred in a maiden lady, more than 40 . The growth was situated on the left side, shape, size, and attachment, with that described er case, but ulcerated in its entire extent, which lso extended to some little distance around its s. The ulceration had been spreading for several the fetid odour it emitted was singularly loath- his I am confident must originally have been

occasioned by the personal filth and disgusting neglect of the patient, who though wealthy, was the most insupportable miser I ever knew, denying herself and servant the most common necessaries of life.

The fetid mass was removed by a simple dissection from the subjacent muscles, as in the former case. Several small arteries were secured after the tumour was removed. A larger surface was from necessity left to be healed by the second intention.

The first four days after the operation were passed without any untoward or unfavorable circumstance. After this she became excessively anxious about her property, accusing her servant and family of robbing her, and saying that she should be left penniless and in want. Fever now set in, which in a few days assumed a typhus form, and soon terminated her life.

Case 3.—A. R, a boy, æt. 14, of a sound constitution, consulted me about a hideous and disgusting deformity of one entire half of his head and face. It consisted of three layers of tumours, from the crown of the head to some distance below the base of the lower jaw. One of them was formed in or involved the eyelids, which were carried down to the lower part of the face. When this portion was raised up the ball of the eye appeared sound in the bottom of a canal three or four inches in depth.

No pain at any time had been connected with this abnormal growth. As the mother states, it was noticed shortly after birth on the side of the face in the form of a small point or pimple, from which all the loathsome and disgusting deformity has proceeded. This boy was so exceedingly deformed and monstrous, as to be an object of terror to the children in his vicinity, and of sport and amusement to the idle boys.

These tumours had the same brawny feel as the rest, and the cuticle had a dry corrugated and scaly appearance where it covered the striæ and irregularities of the hypertrophied tissues underneath. This abnormal mass now involved the

scalp of one side of the head, from the vertex to the centre of the forehead, one half of the nose, the upper and lower lips, the whole side of the face, and extended below the base of the lower jaw, back to beyond the ear. At all these points it seemed to occupy the whole of the dermoid tissue, and was continually increasing in size. (*Vid.* plate II.)

The age at which he had now arrived gave him interest in his personal appearance, and induced him to ask if some change could not be made in it for the better by a surgical operation. This I attempted to do at his request, apprising him and his parents at the same time that the operation was attended with danger.

After being put under the influence of chloroform, an incision was commenced at the vertex of the head, and carried down over the forehead, inner canthus of the orbit, centre of the side of the nose, by the edge of the angle of the mouth, over the chin, and a short distance upon the neck. Another, from the same point above, reached along by the ear, and passed over the base of the jaw, and met the other upon the neck. One or two transverse incisions were afterwards necessary to put the parts in a favorable state to bring the edges together and adjust them by sutures.

The redundant reticular or areolar tissue, was so great, and appeared so completely to occupy the place of the natural structures, that not a muscle of the face could be found after the most careful dissection. It seemed, indeed, as if this growth extended to the membrane of the mouth. The vascularity far exceeded my expectations for such a tissue. A great number of arteries required ligatures. The quantity of blood lost was considerable from the arteries, as also from several large veins. Adhesive plaister, lint, and a double headed roller, completed the dressing.

Shortly after recovering from the anæsthesia, an anodyne of morphine was exhibited to allay pain and quiet the mental agitation under which the boy laboured. Much of the wound healed by the adhesive process, and what suppura-

tion followed seemed to favour the melting down and destruction of the superabundant morbid tissue that remained.

During all the process of granulation and cicatrization, firm pressure was kept up by a roller carefully adjusted to all parts of the head and face.

He recovered completely from the operation, and his improved appearance was greatly calculated to give satisfaction to himself and family, as well as to gratify the operator. It was not many weeks, however, before there were striking evidences of a renewal of the growth, in defiance of the continued pressure of the roller bandage.

The growth was gradual but constant, and after some months its magnitude was so considerable as to induce him again to request its removal. Its size was now far less than at first.

Putting him under the influence of chloroform, several incisions were made about the face and side of the head, in order to remove as much of the morbid part as possible, as well as to secure more points for cicatrization. He lost much less blood in this than the first operation, and comparatively but few arteries required ligatures. No untoward circumstance attended the after treatment, and again much benefit resulted to the patient in appearance.

This improvement, however, was but temporary, for in less than a year the morbid formation commenced again to grow, and now has attained a magnitude, which though less than that of the original growth, makes him a hideous object.

Case iv.—A boy in excellent health, about twelve years old, consulted me about a tumour on the opposite side of the face, identical in all its physical characters, but of less size, than that in the preceding example. As his appearance was far from being beautiful, he was also desirous of being improved by an operation. This I consented to do; preparing him, however, with the probability that it would return. This growth commenced also in early infancy, and, from all that could be learned, was probably congenital.

While in a state of anæsthesia, I made several extensive incisions, and cut more liberally into the surrounding healthy integuments, with a view of not only removing as far as possible all the abnormal tissue, but to obtain also the benefit of the two reparative processes of granulation and cicatrization. The benefit of this soon became very apparent, but the permanency of the cure may be greatly attributed to the persevering use of compressed sponge, and tight bandaging over the granulating surface by my estimable friend, Dr. Batchelder. About six years have now elapsed, and there has not been any return of the morbid formation.

Case v.—This was truly a monstrous morbid production. Though disgusting, and even frightful, to ordinary beholders, there was in its organization and external characters, looking at it as a morbid growth, something symmetrical and beautiful. From the mother's statement it was observed soon after birth.

Miss S—, æt. about 45 years, of robust country health, came to me from the western part of the State of New York, as she said, to show me a tumour, and to know if it could be removed. From the compact and regular arrangement of her dress, the impression on my mind at once was, that it was of no great importance as to size. My astonishment was not a little excited, as soon as she removed her dress from the chest and neck, not only from the immense size of the mass, but that it could all be so completely stowed away, as not to disturb the apparent symmetry and harmony of her proportions.

The tumour was of a dark brown or copper colour, of a soft elastic feel, very much resembling a collapsed lung or a placenta. It hung in beautiful and fantastic folds, like the convolutions of a tippet over the neck, shoulder, and chest. There were five of these folds or stories, the smallest above, the longest or broadest below.

It was attached to the healthy integuments behind and in front of the ear—directly under its lobe—to the entire

extent of the side of the neck from near the nucha to the edge of the larynx and trachea, to the whole line of the clavicle and middle of the upper bone of the sternum, over the shoulder, part of the scapula, and reaching upon the arm to near the insertion of the deltoid muscle—over the entire pectoralis major to the middle of the sternum and ensiform cartilage, and to the upper part of the rectus abdominis and latissimus dorsi, with a portion of the serratus magnus anticus. The lowest loose fold hung a little below a line with the umbilicus. The entire length of the tumour was twenty-one inches, its breadth eighteen inches.

Stating to her that I thought it might be removed, she requested to have the operation performed, if there was, as she said, "any chance of her life." Being made insensible with chloroform, the operation was performed in the following manner:

An incision was made a little below the tumour, across the lower part of the deltoid muscle, and the growth was dissected from this muscle to the top of the shoulder, then from the side of the thorax and upper part of the abdomen, then from the whole line of the clavicle, the upper part of the sternum, the back of the neck, and from the trapezius muscle. It was now detached from about the ear, and the dissection continued towards the front part of the neck, in the direction of the course of the sterno-cleido mastoid muscle, until it terminated by an incision over the mesial line of the larynx and trachea in their entire length.

In all this extensive dissection many arteries required ligatures, and some of them were of considerable size. The most remarkable, and indeed monstrous, were two veins entering the sub-clavian, no doubt the external jugular in its anterior and posterior branches, each apparently separate. Their size was the greatest I ever saw in any superficial veins, being each not less than my fore-finger. They were running close together, and were seized successively the instant on being cut with the forceps, and were tied, to prevent the admission of air, from which, on one occasion, I had seen frightful and almost fatal effects.

As the tumour originated on the upper part of the neck, these two enormous superficial veins were probably the principal channels for returning the blood from the whole abnormal mass. They lay side by side, as they went through the deep cervical fascia, but probably just on entering the subclavian, they united, as is usual in the normal state.

As the operation was considerably protracted, from the extensive superficial dissection, and the large number of arteries which required ligatures, some exhaustion followed, but she was not alarmingly depressed at any moment, and quickly rallied when the anæsthesia was allowed to pass off.

After she had sufficiently recovered for any vessels to show themselves, and all bleeding was stopped, the wound was dressed with dry lint, compresses, and a roller to make a moderate amount of pressure. Shortly after the operation, an anodyne was administered to allay pain, and lessen irritation. For several days afterwards, until suppuration was established, she had considerable fever, but much less than I anticipated from so extensive a wounded surface. The fever was readily moderated by appropriate treatment, and the granulating process was soon established.

After between two and three weeks of progress in healing, everything seeming to be as favorable as could be wished, erysipelas spread itself extensively around the edges of the wound, accompanied by a vitiation of the discharge, and a sweeping away of the healing material. This state was accompanied with great disturbance of the brain and nervous system, with low fever, which imminently hazarded her life. By suitable and appropriate local and general treatment, the storm was arrested, the nervous and vascular system returned to a quiet and tranquil state, the wounded surface assumed a healthy appearance, and all again promised well.

Two or three weeks were now passed in an improving way, but on a sudden, without any physical cause, another attack

of erysipelas burst upon the healthy and healing wound, affecting the general system, and put us all back again. This was, however, less violent than the former attack, and produced less havoc among the granulation. These seizures were from an atmospheric cause, erysipelas being at this season very prevalent in private as well as hospital practice.

By means of general tonics, nutritious diet, and stimulating dressings, the wound soon assumed again a more favorable appearance, and began to granulate and cicatrize. From the immense extent of the ulcerated surface, and the enfeebled state of the general system, various changes in the constitutional and local treatment were from time to time called for. These changes were properly met, and the wound had so far healed in three months that she was enabled to return home—a distance of more than two hundred miles.

On getting to her native village, she rapidly improved; and was soon enabled to return to her accustomed duties.

I have heard several times of her, and within three weeks had a letter from her. The wound for some time was entirely healed; but, at the time of her last letter, there was a small point that had ulcerated, without, however, manifesting any disposition to spread, or any peculiarity in its character. The critical period of life at which she had arrived, indicated to my mind the propriety of establishing an issue somewhere: I accordingly directed one to be made in the sound arm.

There has never been any appearance of a reproduction of the tumour; nor is there any at the present time. It is now about five years since the operation was performed.

This tumour weighed nine pounds, and was twenty-one inches in length, and eighteen in breadth.

My colleague, Professor Lovett, has kindly furnished me with the following notes of the microscopical appearance of one of the tumours:

"The specimen appears to me to consist of an hypertrophy of the skin, and of the subcutaneous cellular tissue.

"Under the microscope I find nothing but an exaggeration of the natural tissues. There are no evidences of a malignant formation.

"The diseased structures seem to me to be quite analogous to what was noticed in the case of elephantiasis of the leg, which I exhibited to you during the winter."

J Plumbet del N Y C.H Ford lith.

Ford & West Imp

J. Phoulet del. N.Y. C.H.Rand. lith.

Pond & West Imp.

CASE
OF
DISTORTION OF THE SPINE,
WITH
OBSERVATIONS ON ROTATION OF THE VERTEBRÆ AS A COMPLICATION OF LATERAL CURVATURE.

BY
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DEMONSTRATOR OF MORBID ANATOMY AT ST. THOMAS'S HOSPITAL.

Received May 9th.—Read June 27th, 1854.

THE late Gideon Mantell was born at Lewes, in the county of Sussex. He applied himself at an early age to the exploration of the fossils of the upper chalk formation, and on coming to London to attend the medical school of Bartholomew's Hospital, he took with him a collection of considerable extent.

On his return to Lewes to engage in practice, he still continued his investigations, and it will easily be believed that his labours were great, inasmuch as his most remarkable and successful researches were carried into the Galt and Wealden rag,—in the hard and untractile masses of the latter he discovered the remains of those lost gigantic reptiles, the Megalosaurus and Iguanodon.

Impaired health and other causes induced him to transfer his practice from Lewes to Brighton, and some time after he again moved, and settled at Clapham, where his personal labours as to his favorite pursuits were restricted to the collection and examination of the few fossils to be met with in the gravelly alluvium.

It appears that in 1842, being 52 years of age, he suffered from excessive pain in the back, inducing him to apply an opiate liniment and leeches. Then he was thrown from his

carriage upon the back; and the symptoms are reported to be aggravated, and numbness came on in the left foot.

After this it is stated that he attended a case of concussion of the brain, and walked home in an intensely cold night, when the lower limbs became paralysed, the bladder required the catheter. The rectum was also affected, and enemata were used.

After many weeks the power of voluntary motion slowly returned; sensation followed with intense neuralgia. The tumour in the back rapidly increased, with supposed fluctuation. During the period of nine months, Liston, Brodie, Bright, Lawrence, Stanley, Coulson, &c., were consulted. The tumour became slowly harder, and almost disappeared. Sensation continuing to return, the tumour again became larger and harder, and the abdominal aorta was pushed forward.

The cachectic appearance of the patient led to the suspicion of the formation of a malignant tumour in connexion with the bodies of the vertebræ, an idea which was subsequently abandoned.

By degrees the neuralgia became less frequent, but Gideon Mantell notes his health as broken up.

To relieve intense suffering, he sometimes resorted to anodynes, but it does not appear that he ever prescribed large doses for himself. On the last occasion a dose of this kind, which is believed to have been taken on an empty stomach, produced the symptoms of narcotic poisoning, which proved fatal.

Report of the Post-mortem Examination of Dr. Mantell. *By* Mr. Wm. Adams.

Died 10th November, 1852. Examination of the body 13th November.

A tall, well-developed, muscular man. As the region of the spine was the chief seat of interest, from there being a history of a tumour or swelling having existed in the left lumbar region, supposed to be dependent upon, or connected with disease of the spine, the body was first placed in

the prone-position, and the dissection commenced from behind.

There was a slight fulness in the left lumbar region near to the spine, but certainly nothing amounting to a tumour. On pressure, three or four hard and prominent nodules could be felt, one above the other, situated from 2 to 3 inches to the left of the spinous processes, and on a level with them.

No lateral deviation of the spinous processes could be detected in any portion of the column. Both sides of the chest were fully and symmetrically developed. No tilting of the pelvis was apparent, though some deviation might have existed, as exact measurements were not taken, nor were the pelvic bones thoroughly exposed by dissection. It may therefore be said that the body did not present any remarkable external appearance. No obvious deformity existed. In pursuing the dissection from behind towards the abdominal cavity, it soon became apparent that the prominent nodules felt in the left lumbar region were the apices of the transverse processes of the lumbar vertebræ projecting backwards, and rising to the level of the spinous processes. The bodies of the lumbar vertebræ could also be felt projecting in an arched form, with the convexity outwards to the left side, and at first suggesting the idea of a hard tumour connected with the vertebral column. No morbid appearance presented itself in the soft tissues in this region. The subcutaneous cellular tissue and fat were everywhere perfectly healthy; the muscles were apparently quite healthy. There was no trace of any morbid growth, cyst of abscess, or of any other inflammatory process having existed in the neighbourhood; no thickening, adhesion, or other alteration in any of the soft tissues.

The body was now placed on the back, and the abdominal cavity was laid open in the usual way. A very severe lateral curvature of the spine to the left side in the lumbar region was now seen. The curved portion of the spine presented also a remarkably twisted appearance, from lateral rotation of the vertebræ, so that the transverse processes of the left

side projected backwards, as above described, whilst those on the right side projected inwards towards the abdominal cavity. The anterior common ligaments of the spine, and the cellular tissue on the anterior and lateral aspects of the vertebræ, and also the psoæ muscles and other tissues, were in a perfectly healthy condition.

No traces of abscess or of any old inflammatory process could be found, and it being impossible that a lumbar abscess could have formed and disappeared without leaving some structural changes in the soft tissues surrounding the spine, it may confidently be asserted that no abscess ever existed.

A portion of the vertebral column, consisting of the three lower dorsal and all the lumbar vertebræ, with a portion of the sacrum, was removed for separate examination. Viewed from its posterior aspect, the apices of the spinous processes of this portion of the spinal column are seen to present a lateral deviation to the left side, the most prominent part of which, viz., between the spinous processes of the second and third lumbar vertebræ, measures rather more than half an inch from a vertical line drawn from the spinous process of the tenth dorsal vertebræ to that of the first sacral bone. This distance is easily diminished to a quarter of an inch by a slight effort at straightening, and this, as above stated, was not apparent previous to its removal, though as measurement was not then adopted, it might have existed.

The bodies of the spinous processes, however, instead of passing directly backwards, incline towards the left side, so that in this aspect, the sides of the spinous processes are brought into view. This deviation exists in the spinous processes of the first, second, third, and fourth lumbar vertebræ, but to a much greater extent in the second and third than in the others, the angle of lateral inclination in these being nearly 45°. The spinous process of the fifth lumbar vertebræ retains its normal direction.

The transverse processes of the first, second, and third lumbar vertebræ on the left side project backwards towards

the skin at about an angle of 45°, and rise exactly to the level of the apices of the spinous processes. The transverse process of the fourth lumbar vertebra is very remarkably altered both in direction and form; instead of passing directly outwards, it curves upwards as a horn-like process, and approaches the transverse process of the third vertebra within three eighths of an inch; and instead of being flattened in its antero-posterior aspects, it is compressed from above downwards, and expanded horizontally towards its free extremity. This expanded portion measures three quarters of an inch in its transverse diameter, and its under surface presents a shallow cup-like depression, coated with a thin layer of fibro-cartilaginous substance, giving to it a well-marked character of an articular surface.—There can be but little doubt that this transverse process rested upon the crest of the ilium, articulating with it, as it were, by its expanded extremity, the form of which, together with its altered direction, being the result of long-continued pressure from the superincumbent weight. The oblique section made through the sacrum, in removing the parts, has unfortunately not included the corresponding portion of the crest of the ilium, but the above supposition is rendered exceedingly probable by the general aspect of the parts, and is also supported by the existence of a similar condition in the specimen closely resembling the present from Mr. Cæsar Hawkins's collection at St. George's Hospital. In this preparation the pelvis is attached, the corresponding transverse process presents a precisely similar appearance, and has only been slightly separated from the crest of the ilium, upon which it obviously rested, in the process of drying. The transverse process of the fifth vertebra has been sawn through in removing the parts, but from the portion still remaining, it must have been either absorbed to a great extent, or remarkably altered in form and position.

The transverse processes on the left side are widely separated from each other, whilst those on the right side are proportionably approximated. The distance between the transverse processes of the first and second lumbar vertebræ

on the left side measures rather more than an inch, and between those of the second and third vertebræ one and a quarter inches. The tranverse processes of the third and fourth vertebræ on the same side are remarkably approximated, in consequence of the altered form and direction of the latter above described; they are only three eighths of an inch apart. The transverse process of the fifth vertebra has been sawn through in detaching this specimen. The distance between the transverse processes of the first and second lumbar vertebræ on the right side is only half an inch; and between those of the second and third vertebræ, and also of the third and fourth vertebræ, rather less than half an inch. The transverse processes of the fourth and fifth vertebræ on this side appear to be abnormally separated from each other, to some extent, the distance between them measuring fully an inch.

The articular processes have evidently been subject to a very severe amount of irregular pressure and strain, tending towards displacement, but they have at the same time become gradually altered in form, and considerably enlarged by the growth of bone, principally at the margins of the articular surfaces, which have thus been retained in contact. These appearances are well seen in a transverse section which has been made of one of these joints. The process by which the enlargement has taken place appears to be similar to that by which the enlargement of the articular extremities of bones (in the hip and knee-joints for example) has been shown by Mr. Wm. Adams to take place in the affection called chronic rheumatic arthritis. (See 'Trans. Path. Soc.,' vol. 3, paper by Mr. Wm. Adams.)

Viewed from its anterior aspect, the specimen exhibits a very severe degree of lateral curvature to the left side, involving the bodies of the two lower dorsal and the three upper lumbar vertebræ, with a remarkable degree of rotation of the vertebræ in the same direction, and also a very perceptible lateral curvature to the right side, involving the bodies of the two lower lumbar vertebræ and the sacrum; so that a distinct double curvature exists. The most pro-

minent point of the upper and larger curve is the intervertebral substance between the second and third lumbar vertebræ. If a vertical line be drawn from the centre of the tenth dorsal vertebra, and carried downwards through the centre of the sacro-lumbar articulation, it will be found, that the distance between this vertical line, and the most prominent part of the lateral curve, viz., the outer border of the intervertebral substance between the second and third lumbar vertebræ, measures three and a half inches.

The deformity of the spine cannot, however, be correctly described as a direct lateral curvature, for the bodies of the first, second, third, and fourth lumbar vertebræ are also rotated in a horizontal or transverse plane towards the left side, so that the anterior surfaces of the bodies of the second and third vertebræ have a lateral, rather than an anterior aspect. The rotation in these vertebræ, extends to very nearly 45° from the median plane. The eleventh and twelfth dorsal, and the first and the fourth lumbar vertebræ, are also implicated in this lateral rotation. In this anterior aspect of the specimen, it is also apparent, that absorption of the bodies of the vertebræ in the concavities of the curves, especially of the second and third lumbar vertebræ, and also of the intervening intervertebral substance in the upper curve, and of the intervertebral substance between the fourth and fifth lumbar vertebræ in the lower curve, has taken place to a considerable extent. In these situations, there is not the slightest indication of any inflammatory process having existed.

Viewed in profile, or from its lateral aspect, the natural curve of the spine, in the lumbar region, is seen to be reversed; and instead of presenting a convexity forwards, the three upper lumbar vertebræ, together with the twelfth dorsal, present anteriorly a concave outline. This is not produced by any absorption or destruction of the bodies of the vertebræ anteriorly, but is evidently caused by the very remarkable degree of rotation above described; by which the natural anterior convexity in this region, is made to

assume a lateral position, as if the spinal column had been laterally twisted on its vertical axis, the centre of motion being fixed at the apices of the spinous processes.

A vertical section, from side to side, through the bodies of the vertebræ, exhibits the following appearances. There are no indications of any destructive disease, such as caries or necrosis, having existed in any of the vertebræ. The cancellous structure appears to be healthy in all parts. The chief alteration in the bones, is a diminution in thickness of the bodies of the vertebræ in the concavity of the larger curve, the result of absorption from unequal pressure; this chiefly affects the bodies of the second and third lumbar vertebræ, each of which is diminished a quarter of an inch on the right or concave side: these vertebræ each measure one and a quarter inch in thickness on the convex, and one on the concave side of the curve. The wedge-shaped form thus given, to a certain extent, is less than might have been expected from the severity of the curve externally.

The intervertebral substances between the bodies of the and first and second, and of the second and third lumbar vertebræ, have been, to a considerable extent, absorbed in the concavity of the curve; these cartilages each measure five eighths of an inch on the convex, and less than a quarter of an inch on the concave side of the curve; this, also, is evidently the result of unequal pressure; there are no indications of ulceration having existed.

It is therefore obvious, that the lateral curvature is chiefly dependent upon absorption of the intervertebral cartilages. The intervertebral substance between the third and fourth lumbar vertebræ, is uniformly diminished in thickness to a quarter of an inch through its central portion, but expanded at each side, where it has been free from pressure, in consequence of a certain amount of lateral sliding, or displacement of the body of the third vertebra from the fourth; the lower border of the third vertebra projects beyond the upper border of the fourth vertebra, three eighths of an inch towards the left or convex side; and on the opposite side

has receded, as it were, from the edge of the fourth vertebra to a like extent. The body of the fourth lumbar vertebra, by a similar movement of lateral displacement, also projects beyond the body of the fifth vertebra three eighths of an inch to the left side.

The intervertebral substances between the fourth and fifth lumbar vertebræ, and between the fifth vertebra and the sacrum, are diminished in thickness, in an opposite direction, to those between the second and third, and the third and fourth lumbar vertebræ. The cartilage between the fourth and fifth vertebræ measures rather less than a quarter of an inch on the left side, and rather more than three eighths of an inch on the right side; so that it is diminished a quarter of an inch on the left side. The cartilage between the fifth vertebra and the sacrum is diminished in the same direction, but to a somewhat less extent. The bodies of the fourth and fifth vertebræ are not diminished in thickness on either side; they both measure five-eighths of an inch in thickness in all parts. The wedge-like form of the last two intervertebral cartilages described will be seen to produce a curvature to the right side, the arc of which would include the bodies of the fourth and fifth lumbar vertebræ and the first bone of the sacrum.

As minor alterations in the osseous structures, indicating a reparative process, may be mentioned a considerable increase of thickness and density of the compact structure forming the outer surface of the bodies of the vertebræ in the concavity of the curve, and also of the adjacent portion of the cancellous tissue similar to the thickening of the walls of the long bones in the concavities of the curves following rickets in early life, and no doubt answering a similar purpose of buttress-like support. The superior and inferior margins of the bodies of the vertebræ are also enlarged so as to form projecting lip-like processes.

It was not considered advisable to lay open the spinal canal for the purpose of examining the condition of the cord, though such a proceeding might appear to be necessary to the explanation of some of the symptoms in this

case. The section for this purpose could not have been made without totally destroying the specimen as one of deformity, and in this respect it presented so many features of interest and practical importance, that their preservation was considered to outweigh the chances of discovering any morbid changes in the spinal cord.

The fact of the greatest practical importance which this specimen illustrates and clearly proves, is one which I believe has not hitherto been described, viz., that a very severe degree of lateral curvature of the spine with transverse rotation of the bodies of the vertebræ, accompanied with lateral absorption of the bones and intervertebral cartilages to a considerable extent, and attended with all the distressing symptoms of the most aggravated form of this affection, may exist, with only a very slight lateral deviation of the apices of the spinous processes; in short, that the severest degree of deformity of the spine may exist internally, without the usual indications in respect of the deviation of the spinous processes externally.

When it is borne in mind that all surgeons are in the habit of relying upon the relative position of the apices of the spinous processes to the median line, as an index to the existence or non-existence of lateral curvature, the importance of the fact above described cannot be over-estimated in the diagnosis of this affection. In this particular case it does not appear that any of the very eminent physicians and surgeons who examined Dr. Mantell suspected the existence of lateral curvature of the spine; the general opinion seems to have been that destructive disease existed either in the bodies of the vertebræ or intervertebral substances and was accompanied by lumbar abscess, which one surgeon proposed to open. The fact, however, that the supposed lumbar abscess made no progress after the lapse of a considerable time, from one to two years, but on the contrary rather diminished, threw considerable doubt and obscurity over the case; still in the absence of the great diagnostic symptom of lateral curvature of the spine, viz., lateral deviation of the apices of the spinous processes, this

affection was not suspected; and it does not appear that the hard nodules felt in the lumbar region, and once supposed to be the lobules of a tumour connected with the bodies of the vertebræ, were at any time recognised as the transverse processes of the vertebræ. This can hardly be matter of surprise, when it is remembered that it was the only positive symptom, taken in conjunction with the general aspect and inclination of the body, by which the affection could have been diagnosed, and up to the present time such a condition has not been described as diagnostic by any authority on curvature of the spine. A careful study of the present case will, however, enable us to diagnose a similar condition in alike case, with as much certainty as if the ordinary indications were present.

The condition of the spine here described as transverse rotation of the vertebræ, the centre of motion corresponding to the apices of the spinous processes, I do not find mentioned by any modern authority on these affections; but it appears to have been observed by the late Dr. Dods, of Bath, who, so far as I know, was the first to direct the attention of the profession to the subject of rotation of the spine in lateral curvature. In the year 1824, he published a somewhat remarkable work, entitled, 'Pathological Observations on the Rotated or Contorted Spine, commonly called, Lateral Curvature.'

The author endeavoured to show, that the condition generally described as lateral curvature, was really one of transverse rotation of the vertebral column, the natural flexures of which were by this movement brought more or less into view posteriorly instead of laterally; that, in fact, as an object becomes changed in its appearance from change of position, or by varying the point of sight, so an altered position, the result of rotation of the spinal column, produces the deceptive appearance of lateral curvature. At page 98, he observes, "It does not happen in all cases of contorted spine that the whole column is moved round; if it were so, we should have invariably the profile of its three flexures brought into view in the manner described, whereas,

it is well known that there are frequently but two of them observed." At page 23, he remarks, "As the spine is rotated spirally, and not as upon a pivot, the profile of its flexures will be imperfect." Dr. Dods appears to have been led to the existence of rotation by observing what was really the most positive symptom in the case now under consideration, viz., the prominence of the transverse processes in the left lumbar region. He states, page 101, "During the course of my operations (alluding to friction, &c.,) upon several patients, I was struck in all of them, (for they were all contracted to the right side,) with a considerable bony hardness and projection on the left side of the loins, raised nearly to a level with the spinous processes; and this I found to be the case in the patients whose spine exhibited little or no apparent curvature in the loins, as well as in those in whom the apparent curvature was very great." After the muscles had been relaxed by friction, Dr. Dods was enabled to satisfy himself that the bony prominences were produced by the transverse processes of the lumbar vertebræ, which could be distinctly felt and counted like the spinous processes. In these cases the transverse processes of the same vertebræ on the opposite side could not be felt, and appeared to have sunk inwards completely out of reach. Reasoning upon these facts, and considering that a direct lateral curvature of the column could only affect the transverse processes by separating them on one side, and approximating them on the other, without altering their transversity with respect to the body, Dr. Dods concluded that such a condition could only be produced by a movement of transverse rotation. He also traced a similar condition in the dorsal region, evidenced by the oblique position of the spinous processes, also described in Dr. Mantill's case, and considers the rotation sometimes to extend to the cervical region.

From the above observations, it would appear that Dr. Dods had met with and recognised the precise conditions now described in the specimen under consideration; for he specially mentions the fact, in some cases, of the transverse

processes rising to the level of the spinous processes in the lumbar region, with "little or no apparent curvature in the loins;" and we have thought the evidence of this fact of sufficient importance to justify his views being brought under the notice of this Society. They are evidently the result of careful and original observation, though the explanation of the phenomena observed, their mode of production, and the indications for, and methods of treatment given, are in many respects erroneous, like the great majority of pathological doctrines tested by the experience of thirty years' scientific inquiry; it would, however, be out of place to advert to these points in the present communication.

In the cases alluded to by Dr. Dods, his attention was probably directed to the existence of rotation in the lumbar vertebræ by prominence of one of the shoulders, and other points of defective symmetry which may or may not have existed in Dr. Mantell during life, though not obvious after death. By some it has been remarked, that "he looked as if he suffered from curvature of the spine." There is no account, however, of any examination having been made with special reference to this point. From an observation made by Sir B. Brodie, in a clinical lecture delivered by him in Dec., 1846, and published in the 'London Medical Gazette,' it would appear that M. Guérin was familiar with the appearances described in the specimen exhibited. Sir B. Brodie observes, "At a very early period, and even before the lateral curvature is very distinct posteriorly, the bodies of the vertebræ are actually twisted to one side. This curious circumstance was pointed out to me by M. Guérin, who has some preparations, in which the fact is very perceptible." In M. Guérin's first memoir, 'On the Treatment of the Deviations of the Spine by Section of the Muscles of the Back,' published in 1843, page 18, he alludes to the modifications of form dependent upon the double influence of vertical displacement caused by lateral flexion, and of horizontal displacement caused by torsion. He was evidently aware of the existence of rotation, but he does not allude to it as at any time coexisting with an absence of

lateral deviation of the apices of the spinous processes, as in the present instance, and therefore it is not mentioned in its most important practical bearing upon the diagnosis of lateral curvature. He had probably noticed what may now be described as the disproportion between the internal and external curvatures, also a most important fact, and one of frequent, if not constant occurrence, in all the more severe forms of lateral curvature of the spine.

Rotation of the vertebræ, or a spirally twisted condition of the vertebral column, as a complication of lateral curvature, is alluded to by many English writers on this affection, but generally only as a passing observation, little or no practical importance being attached to it, and by several of the principal authorities of the present day it is altogether omitted. There can be no doubt, however, of its frequent, if indeed it may not be said, its constant occurrence as a complication of the more severe forms of lateral curvature; and when it exists in any considerable degree, it constitutes one of the chief difficulties of treatment. All the instruments at present so generally used, which make direct lateral pressure on the convexity of the curve, must tend to increase the mischief in such cases, though by their effect in flattening the ribs, this result may not at first sight be apparent.

EXPLANATION OF THE PLATES

Illustrating Mr. Wm. Adams's description of a Case of Distortion of the Spine.

PLATE I.

Anterior view of the Spine of the late Dr. Mantell, *vide* p. 172.

PLATE II.

Posterior view of ditto, *vide* p. 170.

C.H.Ford lith. ad nat.

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Bord. & West. Imp.

ON

GOUT AND RHEUMATISM.

THE DIFFERENTIAL DIAGNOSIS, AND THE NATURE OF THE SO-CALLED RHEUMATIC GOUT.

BY

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In this country, there is no one, I believe, whose opinion would be looked upon as an authority, who holds the doctrine of Chomel, that gout and rheumatism are one and the same disease; still there are very many who, allowing the complete separation of these diseases, in their characteristic forms, yet entertain an idea that one disease is able to merge into the other; and that a morbid condition to which the name of rheumatic gout has been given, is not uncommonly produced—a condition whose name is familiar both to the profession and the public, but of which it is difficult to find a precise description.

Can one disease merge into the other? Can rubeola become scarlatina, or scarlatina rubeola? Doubtless it is not unfrequently difficult to diagnose certain cases of either of these diseases, at any rate, simply from the present condition of the patient; it is not, however, customary to designate such cases by the compound name of rubeolo-scarlatina, or scarlatino-rubeola; for we feel confident that each of these diseases is produced by a special poison, and has its own special pathology, although the symptoms produced by one may occasionally simulate those of the other. It may be possible, but it is certainly extremely uncommon,

for a patient to suffer from the two affections simultaneously: to such a case the compound name above mentioned might be appropriate. So, also, it may be asked, can rheumatism merge into gout, or vice versâ? Has not each of these affections also its own special pathology, and is not the name of rheumatic gout, as generally applied, simply a cover for our want of knowledge of the precise affection under which any given patient may be labouring? I would not for a moment be thought to deny the possibility of a gouty patient becoming affected with rheumatism; but I have no hesitation in affirming, as the result of long experience and attention to the subject, that the disease is extremely uncommon, and that the cases ordinarily designated by that name are not those in which such a double disease is present.

The subject of the diagnosis of these masked cases, is, I consider, one of very great importance—important both as to the prognosis and especially to treatment; it is also a subject of no small difficulty.

To diagnose acute gout, when it occurs for the first time in a rich man, of middle age, and affecting the ball of the great toe only, preceded by dyspeptic symptoms, and accompanied by turgescent veins and œdema of the inflamed part, is a matter of the greatest ease; so, also, to diagnose acute rheumatism in a poor girl, with most of the larger joints inflamed, together with the endo- or pericardium, preceded by rigors, and not accompanied with œdema of the affected joints, is one of no great difficulty, even to a tyro in medicine; but the case becomes altered when either gout or rheumatism has never been very decidedly marked, or when, from repeated attacks, the symptoms have lost all their pristine characteristics.

To clear away the difficulties in making such a diagnosis, to enable the two diseases to be separated when they assume their masked forms, and to show the impossibility of the frequent occurrence of a disease which can correctly be called "rheumatic gout," is the object which I shall endeavour to accomplish in the present communication.

In a paper published in the Society's Transactions for 1848, entitled "Pathological Condition of the Blood in Gout and Rheumatism, &c.," I threw out the following suggestion with regard to the diagnosis of gout and rheumatism: "Might it not, in doubtful cases, be possible to determine the nature of the affection from an examination of the blood?" At that time, the amount of evidence on this point which I was enabled to bring forward was very limited, amounting only to four cases of gout, and the same number of rheumatism. Since that period, although I have not made known any further evidence on the subject, from an unwillingness to form conclusions from few or imperfect data, I have by no means been unmindful of the matter, and have lost no opportunity of putting the question to a most searching investigation; the results of which, founded as they are on 177 examinations of the blood, taken from 148 separate patients, will be given in the present paper.

I have avoided referring here to any case of either gout or rheumatism, when the blood has not been examined, although during the time in which these have been accumulating, very many others have come under my care.

The plan adopted for tabulating the patients, has been to divide the cases into four different classes.

1. Articular affections, in which was demonstrated the *presence* of an abnormal amount of uric acid in the blood.

2. Articular affections, in which the *absence* of uric acid in the blood was shown.

3. Articular affections, proved to be closely connected with *urethral* affection.

4. Affections *non-articular* in character.

The examination of the blood for uric acid, was in general performed in the manner described in my paper read before the Society this session, which I named the "Uric Acid Thread or Fibre Experiment," except in certain cases where the *acid* was separated and weighed, and the results of which are detailed: the history, symptoms, &c., of each patient are taken chiefly from my Hospital Case-Books, during the time I have been attached to the Institution as

physician; some few have been the results obtained from patients in private practice; but, as must be evident to all, on points connected with the condition of the blood, and requiring accurate investigation, no patients offer the same facilities as those residing for the time in the wards of an hospital.

It will be seen, in referring to the following tables, that the blister fluid has occasionally been analysed as well as the blood; sometimes, but very rarely, in lieu of that fluid, from what I have shown in my last communication to the Society, the condition of the blood may be deduced from that of the blister-serum, when certain precautions are taken.

TABLE I.—*Articular Affections—Uric Acid in Blood.*

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
Wm. Fletcher.	38	Gas-fitter, occasionally musician. Has never had lead colic	Drank much gin and beer, or porter.	Father gout or rheumatism. Mother's father gout, and all mother's brothers. Also own brother. See below.	Pretty good. Fractured ribs about 6 years since; since then affected with present disease.	Numerous. 4 or 5 well marked.	Much drinking before present attack.	Commenced in ball of left great toe; after 4 or 5 days, knees, elbows, hands, and fingers, and left ankle. Left metacarpal and phalangeal joints affected. Pitting on pressure of parts. Pulse 92, hard and full; tongue slightly furred; some thirst; deposits of urate of soda on palmar surface of left index finger. Not on ears.	Clot firm; serum alkaline; sp. gr. 1029·4 at 68° F.; much uric acid by thread experiments.
Fred. Plant.	43	Painter (House.)	Drank freely, porter and gin. Not in great excess; 6 pints of porter daily often.	Apparently hereditary from father, who, however, died whenpatient was young.	Good, except when suffering from gout or lead colic.	Numerous. First attack 11 years since, first in ankle, next in great toe.	None assigned for the present.	Commenced in left knee, then dorsum of left hand, right hand, and both feet and ankles, small joints of hands. Pitting on pressure. Pulse 92, rather hard; tongue slightly furred; appetite pretty good. Deposits in both ears. No other deposits seen. No permanent mischief to joints.	Clot slightly buffed and contracted; serum alkaline; sp. gr. 1028·0 at 60° F.; abundance of uric acid. *Blister fluid* from inflamed joint; no trace of uric acid. *Blister fluid* from abdomen gave a moderate amount of uric acid.

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
Jos. Elliot.	64	Stonemason.	Not stated.	Not stated.	Good.	Numerous. First attack occurred 20 years since in some part of foot.	Not stated.	Affection of hands, ankles, knees, hips. Pulse 80. Deposits in ears; and little nodules in integuments near eyes.	Uric acid in serum.
Wm. Finch.	40	Wine-cooper	Has drunk freely of beer and gin.	Father and grandfather on same side had gout, and father had chalk stones.	Pretty good.	Numerous. First about 10 years since in feet and ankles.	Patient had often brought on attacks by the use of port wine.	Commenced in knee, then elbow, and small joints of hands. Pitting on pressure. Pulse 76. No deposits of urate of soda noticed.	Serum; considerable quantity of uric acid.
Chas. Farr.	38	Brewer's Man.	Has drunk very freely, chiefly porter; some gin.	Father's brother had gout.	Good, in general.	Not known; numerous. About 10 years since had first attack, confined to foot.	None assigned.	Both feet, knees, hips, small joints in hands, and in great toe. Pitting on pressure. Pulse 80. No thirst. Minute concretions of urate of soda in left ear. Large semi fluid collection in left foot of creamy urate of soda; some also on right little finger.	Clot firm; buffed and cupped; serum alkaline; sp. gr. 1029·0 at 55° F.; abundance of uric acid. *Blister fluid* from inflamed part, no trace of uric acid. *Blister fluid* from ankle, a few cystals of uric acid.
Chas. Fletcher.	35	Painter.	Temperate; about 2 pints of porter daily; formerly took spirits also.	See under, W. Fletcher, the brother of the present one.	Pretty good, except from present disease.	Second attack. First 1¼ year since, commenced in left foot.	Injury. Struck by shaft; and fracture of ribs, and pleuritis.	Commenced 2 days after injury, in left elbow and fingers; afterwards both feet and right knee, and right index finger. Pitting on pressure. Pulse 108.	Clot normal; serum alkaline; sp. gr. 1026·8 at 50° F. Abundance of uric acid. *Blister fluid* from chest, a mo-

								Tongue furred; thirst. Fluid from right index finger (middle joint) obtained by puncture; milky from urate of soda (4 days after it was inflamed); concretions afterwards appeared on right ear, and in left middle finger.	derate amount of uric acid.
Thos. Bugg.	54	Farrier.	Has always been a free drinker—beer and gin.	Father had gout, also one brother.	Good.	Has had numerous attacks. First attack in feet 7 or 8 years since; does not remember whether the great toe was chiefly affected. Great toes have been specially affected in some attacks.	Injury from kick of horse on chest.	About 48 hours after injury. Dorsum of right hand and foot, and left elbow, upon which he fell. Pitting on pressure. Pulse 80, full, hard; some thirst; appetite good. No deposits of urates.	Clot slightly buffed; serum alkaline; sp. gr. 1026·64 at 58° F. A good sprinkling of uric acid on thread.
John Howel.	45	Publican for many years, formerly a post-boy.	Intemperate Drank much beer and gin. Good diet.	Father had gout, also brothers.	Good, except gouty.	Numerous. First about 14 years since, in right great toe.	Drink.	Ankles and feet chiefly affected. Pitting freely on pressure. One knee also much swollen; tongue clean; appetite good; no thirst. Pulse 76, not resisting. Nodules of urate of soda on both ears.	Clot normal; serum alkaline; sp. gr. 1029·6 at 47° F. Abundance of uric acid by thread experiment.

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
Sam. Norton.	68	Wheelwright.	Regular.	Not known. (Patient very deaf indeed.)	Pretty good.	About the 12th. First attack in ball of left great toe, next in right great toe, afterwards in large joints also.	Not given.	Great toes both affected; left knee slightly; patient has eczema of both legs; rather chronic. Pulse 92. Appetite good. No deposits of urate of soda.	Clot normal; serum alkaline; sp. gr. 1026·0 at 65° F.; considerable sprinkling of uric acid on thread.
J. Z. Rumsey.	48	Stableman, formerly a soldier.	Lived freely, both in meat and drink.	None to gout or rheumatism.	Good.	About 3d attack. About 20 years since, swelling of ball of great toe; next attack commenced also in great toe.	Not assigned.	Commenced in right knee, hip, shoulders, hands, left knee, balls of both great toes. Pulse 110, rather hard; tongue white, furred. Pitting on pressure. No deposits of urate of soda on any part of body.	Clot normal; serum alkaline; sp. gr. 1028·0 at 60° F. A few crystals of uric acid. *Blister fluid* gave crystals of uric acid. *Blister fluid* from inflamed knee no crystals of uric acid. Second bleeding 2 days after: Clot not buffed or cupped; serum alkaline; sp. gr. 1027·1 at 65° F. Pretty good quantity of uric acid. Third bleeding 8 days after: Clot firm; fibrinous coat; serum gave a moderate amount of uric acid by uric acid thread experiment.

Geo. Hugget.	56	Traveller.	Temperate, but has drank much porter; ½ gallon or more per diem.	Not known.	Good.	About 6th attack. First attack confined to right great toe, 10 years since. Other joints afterwards affected.	Was exposed much to cold before present attack.	Commenced in right foot, then left foot, right knee, and hand; left shoulder and hand. Pitting on pressure. Pulse 108, hard; tongue white, furred. No deposits of urates.	Serum gave abundance of uric acid.
George Hull.	52	Cab-driver and proprietor.		No gout or rheumatism from father or mother; but uncles and aunts on both sides subject to gout.	Good, except gout.	Numerous. First attack 30 years since in feet; then second attack in ball of great toes (both). Knees did not become affected until 2 years since; and 1 year since, upper extremities.	None assigned.	Affection of left elbow, wrist, and metacarpal joint of index finger. No deposits of urates. Pulse 86; tongue furred; no appetite. Pitting on pressure.	Clot buffed, firm; serum alkaline; sp. gr. 1025·56 at 64° F. Pretty good sprinkling of uric acid by thread experiment.
Jn. Steedman.	61	Pianoforte-maker.	Lived well; drank moderately of beer and some gin.	None known to gout or rheumatism.	Good, except occasional gout, and latterly some albuminuria.	First attack of gout about 20 years since.	Present attack came on after chest affection.	First examination of blood some little time before the joints became affected. Patient then suffering from chest symptoms and some œdema. Second examination after attack had completely passed off. During attack,	First examination: Clot normal; serum alkaline; sp. gr. 1021·6 at 66° F. A good quantity of uric acid; thread studded with crystals. Second examina-

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
								the right great toe and knee were affected. Pitting on pressure. Had albuminuria, gradually lessening to a mere trace.	tion: Clot normal; serum alkaline; sp. gr. 1022·4 at 66° F. A very few crystals of uric acid.
Chas. Heath.	46	Coachman until 3 years, since which a cabman.	Has drunk freely of beer and gin.	None to gout or rheumatism.	Good, except subject to attacks like the present one.	Numerous. First attack about 20 years since in right great toe and instep only. Second attack 16 years since in same part. Third, 12 years since in left great toe. In subsequent attacks knees and elbows. Attacks gradually increasing in frequency.	None assigned.	Commenced 13 weeks before admission; partially recovered; for 3 weeks has kept his bed. Hands, knuckles, knees and feet; shoulders. Pulse 72, full, hard. No deposits of urate of soda. The first examination of the blood was made during febrile disturbance of the system; the second when the patient had pain in joints, but no fever.	First examination: Clot firm, buffed; serum; sp. gr. 1026·0 at 66° F. No uric acid found by thread experiment. Second examination: Clot normal; serum alkaline; sp. gr. 1024·8 at 65° F. A moderate amount of uric acid.
Jas. Bunyon.	60	Excavator.	Drinks freely.	None discovered	Good.	4th attack. First com-	Appears to have had	Dorsum of left wrist, hand, metacarpal and seve-	Clot buffed, firm; serum clear, alka-

				to gout or rheumatism.		menced in right great toe.	attacks every one or two years.	ral phalangeal joints, and swelling of right side of face. Pitting of parts on pressure. Pulse 72. No deposits of urate of soda in any part of body.	line; uric acid thread experiment gave abundance of uric acid.
Fr. Westcott.	47	Gentleman's servant.	Lived pretty freely. Drank beer chiefly.	None discovered clearly to gout or rheumatism.	Pretty good, except from gout.	Numerous. Not known. Early attacks commenced in great toe.	Cold. Taking hard beer.	Pains of fingers, wrist, elbow, and shoulder of left side; then same on right side; left great toe, right knee. Pulse 104, rather full and sharp. Some thirst, moderate appetite. Pitting on pressure. Desquamation of cuticle. No deposits on body of urate of soda.	Serum; gave by uric acid thread experiment uric acid on thread; serum of blood from loins by cupping gave also the same.
Jas. Quinland.	58	Has been for a long time brewer's drayman.	Drank very freely of beer and gin.	None distinctly discoverable.	Pretty good until the last few years.	Numerous.	Drink brings on an attack.	Left hand and arm, and knees slightly affected. Pulse 80. Pitting on pressure. Several small deposits of urate of soda in ears; and much distortion of small joints of feet and hands.	Clot not buffed; serum clear, alkaline; sp. gr. 1027·5 at 54° F.; uric acid thread experiment gave much uric on thread. About 0·07 grain collected by analysis from 1000 grains of serum.
Mr. C——r.	About 60	Tradesman.	Drank freely, beer, &c.	Not known.	Good, until gout appeared.	Very numerous indeed.	The present attack seemed to be brought on by mental anxiety.	Present attack chiefly in head; delirium, heat of head, &c. Many of the smaller joints of feet and hands much distorted. No distinct deposits of urates.	Blood by cupping from loins; clot firm; serum clear; sp. gr. 1029·2 at 70° F.; uric acid thread exp. gave abundance of uric acid.

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
Thomas May. April, 1854.	53	Cook.	Eats freely of meat; has drunk pretty freely of beer. Not intemperate.	None to gout or rheumatism known.	Good, except when gouty.	Almost innumerable. First commenced in right great toe about 20 years since. For some time the disease attacked great toes only.	None assigned for any one attack.	This patient has had at different times all the joints of the body acutely inflamed, sometimes the larger, sometimes the smaller, chiefly affected in the attack. There is always much pitting on pressure. The pulse, during acute attacks, becomes quick and hard. Nodules of urate of soda on both ears; and chalk stones on many parts of body, both upper and lower extremities. Sometimes discharges of this urate, and formation of abscesses.	Clot buffed, and slightly cupped; serum clear, alkaline; sp. gr. 1027·12 at 60° F. By uric acid thread experiment a considerable amount of uric acid was obtained.
Feb., 1850.									Blood by cupping from loins; serum clear, alkaline; sp. gr. 1026·0 at 53° F. Uric acid thread experiment gave abundance of uric acid.
Nov., 1849.									Clot firm and large; serum clear, alkaline; sp. gr. 1029·1 at 50° F. By uric acid thread experiment an abundance of uric acid, from 1000 grains 0·04 grain collected.

George Roos.	31	Cabman for many years.	Drinks freely of beer, some spirits.	None to gout or rheumatism.	Good before present affection.	About 6; has had joint affection without fever. About 8 years since had a joint affection, like acute rheumatism.		No swelling of joints. Symptoms, subjective only. Appetite good. No thirst. Pulse 70. Complains of pains in joints, and stiffness in walking, which he has had several months.	Clot not buffed, not very firm; serum alkaline; sp. gr. 1027·4 at 63° F. A moderate amount of uric acid.
Wm. Mitchell.	52	Printer.	Before having gout, drank rather freely of gin, some beer; lived well.	None known; but one younger brother has gout.	Good until 14 years since.	Very numerous. First attacked right great toe, about 12 years since. Second attack a year after, great toe also. Upper extremities have not been affected for more than 6 years, and the concretions not more than 3 years.	Not assigned for any one attack.	At different times, different joints are chiefly affected, sometimes nearly all together. Great toes, and smaller joints of fingers, as well as large joints. Patient covered with concretions of urates of soda; many in ears; much crippled.	March, 1853.—Acute attack; clot cupped and buffed; serum alkaline; sp. gr. 1026·4 at 51° F. Abundance of uric acid, shown by uric acid thread experiment. July, 1852.—Acute attack; clot firm, buffed; serum alkaline; sp. gr. 1026·. Gave uric acid in abundance on thread. Jan. 1851.-Acute attack. Abundance of uric acid in blood.
H.——.	50	Painter.	Irregular.	None known	Good until he had painter's colic.	Numerous. First attack about 3 or 4 years since, in feet.	Not assigned. Attack clearly induced by drink.	Present attack affects chiefly joints of hands; but knees and feet stiff and painful. Concretions in ears and around some joints.	Blood serum contains abundance of uric acid.

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
James Tousett.	42	Ginger-beer maker.	Takes beer freely, sometimes a little gin, but not intemperate.	None known to gout or rheumatism.	Pretty good, except gout.	Numerous. First attack about 10 years, very frequent within last 5 years. Not noticed in history which joint was affected in first attack.	A glass of rum will induce an attack. Over fatigue appeared to have brought on the present.	Commenced in hands, than knees, ankles, &c., became affected. Deposits of urate of soda in ears only.	Clot large and firm, buffed; serum alkaline; sp. gr. 1027·2 at 60° F. 1000 grs. of serum gave 0·044 grs. of uric acid.
W. Buttsworthy.	52	Coachman in gentlemen's families for many years.	Drank pretty freely at one time chiefly wine.	None known	Good, until first attack of gout.	Very numerous, had suffered for many years. Came on first in great toe.	Any depressing cause, or drink, would induce fresh attack.	Scarcely ever free from some acute affection in one or more joints. Concretions in hands, feet, elbows, &c., and ears. Gouty abscesses generally discharging more or less creamy urate of soda.	Clot normal; serum alkaline; . sp. gr. 1030·0 at 60° F. 1000 grs. of serum gave of uric acid 0·11 grain.
— Martin.	41	Porter; formerly in army.	Regular.	None to gout or rheumatism.	Pretty good, had some injury from poisoned wound, a few years since.	Third attack	Not known.	Commenced at midnight, in the ball of left great toe. Pulse 100, rather hard. Tongue furred. Appetite pretty good. Afterwards desquamation of skin over ball of toe. No deposits of urates on any part of body.	Clot normal; serum alkaline; sp. gr. 1026·0 at 70° F. Abundance of uric acid by thread experiment. 2d. Bleeding, attack passed off; clot normal; serum alkaline; sp.

									gr. 1024·0 at 53° F. A few crystals of uric acid.
William Riley.	32	House painter.	Diet good; not intemperate, but drinks beer.	Father had gout, brother suffers also from gout.	Repeatedly had lead colic. Gout for 6 or 7 years; commenced in ball of great toe.	Numerous. For 7 months has not been free from gout.	Subject to draughts.	Right foot, great toe, both ankles, knees, right wrist, middle finger of left hand. Tongue clean. No thirst. Appetite good. Pulse 95. Much pitting of swollen joints. No deposits on any part of body. No permanent distortion of joints.	Clot rather cupped; serum alkaline; sp. gr. 1028·0 at 41° F. Abundance of uric acid by thread experiment. *Blister serum*, on partial recovery. Moderate amount of uric acid. Second bleeding, clot normal. Moderate amount of uric acid.
Wm. Brooker.	46	Coal porter	Good living; a considerable amount of porter per diem.	Father affected with gout.	Good.	First attack. Re-attack about 3 weeks after.	Had much night work lately, had been much exposed to cold.	Commenced at night, in ball of left great toe, then outer side of ankle. Considerable pitting on pressure. Pulse slow. Appetite pretty good. No deposits of urates.	Serum gave much uric acid by thread experiment. Clot normal; serum, sp. gr. 1028·6 at 52° F.
						Second attack.		Same parts affected as before, and right great toe in addition.	Serum of blood gave much uric acid by thread experiment.
John Day.	27	Farrier.	Regular. Diet good; takes a considerable quantity of	None to gout or rheumatism.	Good.	First attack.	Exposed to changes of temperature at work.	Commenced in right ankle, then left wrist, right knee and right wrist; same affection of ball of right great toe. Pulse 64. No pitting	Clot normal; serum alkaline; sp. gr. 1027·6 at 55° F. Abundance of uric acid. *Blister fluid*

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
			beer, and some gin.					noticed in notes. No deposits on any part of body. Joint affection *immediately* procured when Colchicum was administered. Prior treatment gave no relief.	clear, no coagula. No uric acid. Small quantity only procured, and that from an inflamed joint.
J. Channon.	30	House painter for 10 years, before a plumber.	Irregular; has drunk much. Porter and spirits.	One grandfather, and father subject to gout.	Not good from frequent syphilitic affections, and gout and painter's colic.	Ninth or tenth attack. First commenced in great toe.	Attacks brought on distinctly by drink.	Toes, ankles, fingers, and wrists. Desquamation of cuticle. Pitting on pressure. Pulse 66. Appetite bad. Blue line on gums. A small deposit of urate of soda in left ear; some distortion of joints.	Clot buffed, not cupped; serum alkaline; sp. gravity 1026·4 at 49° F. Abundance of uric acid by uric acid thread experiment.
Benjamin Fox.	38	Bricklayer.	Pretty regular; but drinks freely of gin and beer.	None to gout or rheumatism.	Good.	Fifth or Sixth. First, 2 years since, commenced in great toe.	Patient thinks exposure to wet and cold.	Commenced in left knee and foot, then right great toe, then both hands, especially index finger of left. Pitting of parts on pressure, and desquamation of cuticle. Pulse 96, sharp. Appetite good. No deposits of urates on any part of body.	Serum clear; sp. gr. 1027. Abundance of uric acid by thread experiment.
Henry Clubb.	51	Grocer.	Regular.	Not known. Not noticed in case books.	Suffers from bronchitis and emphysema, and from occasional attacks of gout.	Numerous.	After an attack of bronchitis which it greatly relieved.	Commenced in ball of left great toe, afterwards left knee. Pitting on pressure. Pulse 108, sharp. No deposits of urates on any part of body. On second bleeding, patient	Clot normal; serum alkaline; sp. gr. 1029·2 at 52° F. Thread well sprinkled with uric acid. A former bleeding gave also much uric

								suffered from dyspeptic symptoms.	acid just before joint affection appeared.

FEMALES.

Eliz. Wells.	51	Laudress; a widow.	Regular.	Father had same joint affection in knees and feet. One brother also had joint affection, and white deposits with crippled limbs.	Good, till within last 10 years.	Numerous. First attack about 10 years since; knees were affected.	None assigned.	Commenced in knees, then ankles, elbows, wrists, and fingers. Pulse 92, rather hard. No deposits of urate of soda in body.	Clot firm, buffed; serum alkaline; sp. gr. 1028·0 at 65° F. A considerable amount of uric acid by thread experiments.
,,	,,					Another attack some months afterwards.		Affecting the left index finger first, then wrist and shoulders, chiefly small joints of right hand; left knee and instep. Desquamation of cuticle. No deposits of urate of soda in body.	Clot firm, slightly buffed; serum alkaline; sp. gr. 1029·2 at 46° F. Much uric acid by uric acid thread experiment. Abundance of uric acid found in *serum from blister*, by uric acid thread experiment.
Mart. Johnson.	53	Servant.	Regular, but has lived rather hard at times.	Mother had gout and chalk stones.	Not very good. Phthisical. Died a few	First attack.	Not assigned.	Commenced about six weeks ago, in ball of both great toes, afterwards knee, when bled, joint disease	Clot cupped and buffed; serum alkaline; sp. gr. 1026·8 at 60° F. A mode-

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
				months afterwards from chest disease.				was not at all intense. Pitting on pressure. No concretions of urates of soda.	rate amount (small) of uric acid. *Blister serum* alkaline; sp. gr. 1024·0 at 65° F. A few crystals of uric acid. Second bleeding, clot buffed and cupped; serum alkaline; sp. gr. 1027·8 at 62° F. A pretty good sprinkling of uric acid on thread.

Name.	Sex.	Account of Patient.	State of Blood.
C. Bowers.	M.	Attack present about 3 weeks. Undoubted gouty patient. No concretions noticed.	Serum alkaline; sp. gr. 1031·0 at 60° F. 1000 grains gave of uric acid, gr. 0·05.
W. May.	M.	Acute attack. Chronic disease of long standing. Always relieved by colchicum. Concretions in ears and joints.	Clot firm, not buffed; serum alkaline; sp. gr. 1027·8 at 66° F. 1000 grains gave of uric acid 0·062 grain.
U. N—.	M.	A patient with gout of great toe and foot.	Serum alkaline; sp. gr. 1031·5 at 60° F. 1000 grains gave of uric acid 0·044 grain.
— Norton.	M.	Patient with great toe gout.	A small quantity of blood taken. Gave abundance of crystals of uric acid.
Thos. Price.	M.	A painter (wrists and hands affected), gout for 10 years. Last attack one year since.	Serum alkaline; 1000 grains gave of uric acid 0·05 grain.
Thos. Hickson.	M.	Gout. Attack now in right hand. Several deposits of urate of soda in both ears. Œdema of parts.	Clot firm, buffed; serum alkaline; sp. gr. 1028·2 at 70° F. Abundance of uric acid.
M. H—.	M.	Patient diagnosed to be gout by a physician.	Clot firm, buffed; serum alkaline. Gave much uric acid by uric acid thread experiment.
— Hall.	M.	Gout for many years. Large joints now chiefly affected. Has had great toe affection. Deposits in both ears; but not elsewhere noticed.	Serum of blood contained much uric acid.
C. H—.	M.	A patient who has suffered many years from undoubted gouty attacks, bled on account of retrocedence of the affections.	Serum alkaline; sp. gr. 1030·1 at 45° F. Gave much uric acid, by uric acid thread experiment.
E. P—.	M.	Disease diagnosed as true gout, by a physician. Attack disappearing rapidly.	Serum gave uric acid crystals on thread. Not numerous.
D. P—.	M.	Acute attack of gout; the first attack, in great toe for some days.	Serum alkaline; gave on thread abundance of uric acid.
C. S—.	M.	Chronic gout. Great toe often alone affected. No deposits of urates on body.	Blister serum gave uric acid on thread, by uric acid thread experiment.
J. S—.	M.	Painter. Gout of great toe, of 2 days' duration.	Clot firm, buffed; serum alkaline; sp. gr. 1028·0 at 65° F. Abundance of uric acid by thread experiment.
— Pavier.	M.	Gout, with many concretions in ears, and around joints. He died from fracture of femur.	Blood taken after death. Serum by uric acid thread experiment gave much uric acid.

TABLE II.—*Articular Affections—No Uric Acid in Blood.*

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
Geo. Jones.	30	Cabman.	Regular.	None to rheumatism or gout.	Good till he had rheumatism.	Third attack. First about 8 years since.	Cold.	Commenced in knees, afterwards shoulder, elbow. Skin perspires freely. Pulse 72. Tongue white, furred; thirst moderate; no appetite. Slight pericarditis; old heart affection.	Clot firm, slightly buffed; serum alkaline; sp. gr. 1027·04 at 60° F. No trace of uric acid.
Jackson Jones.	31	Linen-draper's man.	Regular.	—	Pretty good.	Second attack. First 3 years before.	None assigned.	Commenced in feet, then in knees, wrists, and ankles. Pulse 112, full, hard. Tongue furred; no appetite; thirst. No cardiac affection.	Clot firm; a little cupped; serum alkaline; sp. gravity 1026·8 at 69° F. No trace of uric acid.
Wm. Kell.	20	Carpenter.	Regular. Temperate.	None to gout or rheumatism.	Good.	First attack.	None, except some draught.	Commenced in knees; then wrists, ankles, and feet. Pulse 84, hard; tongue furred; skin hot. No cardiac affection.	Clot normal; serum alkaline; sp. gr. 1028·0 at 63° F. No uric acid.
Thos. Ware.	33	Tailor.	Usually not intemperate. Lives well.	Mother subject to rheumatism; sisters and brothers not affected.	Good.	First attack.	—	Commenced in right shoulder, then left; afterwards right knee. Joints not very acutely affected. Pulse 88, hard; tongue not furred; appetite moderate. No heart affection.	Clot buffed; not cupped; serum alkaline; sp. gravity 1028·4 at 55° F. No uric acid.

Henry Webb.	28	Costermonger.	Not given.	None stated.	Good.	First attack.	Cold.	Commenced in hip, then left ankle, knees; afterwards wrists and hands. Pulse 96, hard; tongue furred; thirst; no appetite. No pitting on pressure. Free perspiration. No heart affection.	Clot firm; serum alkaline; sp. gravity 1028·8 at 48° F. No uric acid.
Joseph Smith.	57	Carpenter.	Lives well; not intemperate; drinks beer, and a little gin.	Grandfather on father's side affected with rheumatism.	Good.	First attack.	Cold.	Commenced in left knee and thigh, then right foot and instep; afterwards right elbow. Pitting on outer side of foot. Tongue furred. Pulse 78. No heart affection.	Clot normal; serum alkaline; sp. gr. 1029·2 at 51° F. No uric acid.—*Blister fluid.* No uric acid.
J. Goodhall.	26	Labourer, lately a potman in a public-house	Not stated.	None to gout or rheumatism.	Not good for the last few years; scrofulous knee, &c.	First attack.	—	Commenced in right thigh, then knee, back, shoulders, left elbow, wrist, and hand. No pitting of dorsum of hand. Pulse 108, rather hard. Tongue furred; perspiration; thirst. No heart affection.	Clot slightly buffed and cupped; serum alkaline; sp. gr. 1030·4 at 47° F. No uric acid. 2d. Clot very slight buffed; serum alkaline; sp. gr. 1030·4 at 42° F. No uric acid.
Jas. Aldrich.	34	Cabman.	Not stated.	Not stated.	Good till he had rheumatism.	Second attack. First attack 8 years since; many joints affected.	—	Commenced in right hand, then feet, knees, shoulders, and hips and ankles, and left hand; also fingers. Pulse 108, hard; tongue furred; thirst; no appetite. No cardiac affection.	Clot little buffed, firm; serum alkaline; sp. gr. 1028·0 at 52° F. No trace of uric acid.

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
Chas. Garlick.	30	Carpenter.	Regular.	None to gout or rheumatism.	Good.	First attack.	—	Commenced in different joints of lower then upper; both wrists much swollen. No heart affection. Pulse 100, hard. Tongue furred; perspires much; thirst.	Clot buffed, cupped; serum alkaline; sp. gr. 1028·0 at 72° F. No trace of uric acid.
FEMALES.									
Harriet Miller.	27	Servant.	Regular.	Father had rheumatism, (not acute.)	About a year since had slight hemiplegia, and six years ago paralysis of right side of face.	First attack.	Cold.	Commenced with tonsillitis; after some days right foot and ankles, then knees, then wrists and shoulders. Slight pericarditis. No pitting on pressure. Pulse 120; tongue furred; thirst; no appetite. Perspires freely.	Clot firm, slightly buffed; much cupped; serum alkaline; sp. gr. 1026·7 at 63° F. No trace of uric acid.
Ann Mason.	32	Dressmaker.	Regular.	None to gout or rheumatism.	Pretty good until lately.	Second attack. First attack commenced about 4 months ago. Second attack had been neglected for many weeks.	Cold.	1st. Commenced about 4 months since, as an attack of acute rheumatism, with nearly all joints affected, and some heart affection. (Not in hospital.) 2d. All joints affected as before; after 8 weeks came into hospital, with swelling of wrists, and dorsum of hands, and some small joints of fingers, knees ten-	Clot not buffed or cupped; serum alkaline; sp. gr. 1030·0 at 60° F. No trace of uric acid. *Serum of blister* clear, pale-yellow; sp. gr. 1026·0 at 60° F. No uric acid.

								der and left elbow, shoulder somewhat painful. Pulse 84; not very febrile.	
Elizabeth Ash.	40	Married.	Regular.	None to rheumatism or gout.	Good.	Second attack. First attack 14 years since, all joints affected.	Cold—wet.	Commenced in shoulders, then ankles; afterwards elbows, wrists, knees. No heart affection. Pulse 105, hard; tongue furred.	Blood serum by thread experiment. No trace of uric acid.
Rebecca King.	21	Servant.	Regular.	Not stated.	Good.	Second attack. First attack 9 years since.	Not given.	Commenced in left foot and ankle, then right hand, wrist, and fingers; afterwards knees. Pulse 100, hard; tongue furred; perspiration; thirst. No marked heart affection.	Clot firm, slight buffed, and cupped; serum alkaline; sp. gr. 1028·8 at 60° F. No trace of uric acid.
S. Sturgeon.	17	Servant.	Regular.	None to gout or rheumatism.	Good.	First attack.	Cold.	Commenced in knees, then almost all joints of body; left wrist, right knee and ankle especially so. Pulse 130, hard; tongue furred; much perspiration. Endocarditis; pericarditis.	Clot buffed, slightly cupped; serum alkaline; sp. gr. 1024·08 at 40° F. No uric acid.
Sar. Southcott.	26	Married.	Regular.	Mother slightly rheumatic; one brother has had three attacks of rheumatism.	Good until lately.	First attack.	Not known; perhaps cold from washing.	Commenced in knees, then ankles and feet; afterwards hands, elbows, and shoulders. No heart affection. Pitting on pressure of dorsum of hand. Pulse 108, hard; tongue furred; moderate perspiration; thirst, &c.	Clot firm, slightly cupped and buffed; serum alkaline; sp. gr. 1027·6 at 48° F. No trace of uric acid.

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
Cath. Lockyer.	25	Married; a laundress.	Regular.	None to gout or rheumatism.	Not very good.	First attack.	Cold and damp.	Commenced in ankles, hips, and knees, then in joints of upper extremities. No heart affection. Pulse 116, hard; tongue furred; thirst; no appetite.	Clot firm, cupped, buffed; serum alkaline; sp. gravity 1031·6 at 69° F. No trace of uric acid.
M. A. Porter.	25	Laundress; for 5 weeks in service before.	Regular.	None to rheumatism or gout; or questionable to rheumatism from father.	Good.	First attack.	Cold.	Commenced in shoulders, then loins, feet, and hips. Since admission, ankles, knees, elbows, wrists acutely inflamed. Pulse 130, not full; pain increased at night; much acid perspiration; tongue furred; no appetite. Pericarditis with effusion.	Clot firm, buffed, rather cupped; serum alkaline; sp. gravity 1029·5 at 60° F. No trace of uric acid by thread experiment.
Ann Parkinson	54	Cook; widow.	Regular.	None to rheumatism or gout.	Good.	First attack. Perhaps one 30 years before, but doubtful. Said to arise from mercurial ointment on hands.	—	Commenced in hip, then in knees, left wrist, and dorsum of hand. Little perspiration. Pitting of hand and legs on pressure. Albuminuria when admitted. Peri- and endo-carditis. Query if very recent.	Clot firm, buffed, rather cupped; serum alkaline; sp. gr. 1024·8 at 60° F. No trace of uric acid.
Mary Ann Robinson.	17	Servant.	Regular.	None to rheumatism or gout.	Not very good. Weak and hysterical.	First attack.	Cold.	Commenced in both ankles, then both knees, hips, shoulders, elbows, wrists, and hands. No pitting on	Clot firm, normal; serum alkaline; sp. gravity 1026·32 at 65° F.

								pressure. Pulse 120, hard; tongue furred, white; much thirst; no appetite. Distinct endocarditis.	No trace of uric acid.
Ann Clarke.	30	Servant.	Regular.	None to rheumatism or gout.	Good.	First attack.	Cold in the feet.	Commenced in knees; heart early affected; then hips and shoulders. Tongue much furred; much thirst; no appetite. Pulse 110 when admitted. Aortic regurgitant in character. Perspiration moderate. Endo- and peri-carditis very severe.	Clot firm, somewhat buffed; serum alkaline; sp. gr. 1026·65 at 60° F. No trace of uric acid.
L. Emherson.	31	Married. Household work.	Regular.	To rheumatism on father and mother's side.	Pretty good. Subject at one time to fainting (hysteria?)	Third attack.	None assigned.	Affection of knees, ankles, wrists, shoulders. Moderate perspiration. Pulse 96, moderate, full; tongue brown, furred; thirst; no appetite. Slight endocarditis.	Clot firm, slightly buffed and cupped; serum alkaline; sp. gr. 1028·5 at 60° F. No trace of uric acid.
Eliz. Smith.	27	Married.	Regular.	To rheumatism from mother. Two sisters affected.	For last few years has been in a weak state of health.	First attack.	Cold and damp.	Commenced in knee, then ankles, hips, shoulders, elbows, and dorsum of hands. No pitting. Pulse 105; tongue furred; thirst; no appetite; perspiration moderate. No heart affection.	Clot firm, buffed, cupped; serum alkaline; sp. gravity 1025·0 at 60° F. No trace of uric acid.
Eliza Wiggins.	43	Married. Takes in washing.	Regular.	To rheumatism from father. One sister affected.	Good.	Second attack. First at 16 years of age.	None assigned.	Commenced in feet, then knees, hips, one wrist, shoulder, muscles of one side of neck and head:	Clot buffed, cupped; serum alkaline; sp. gravity 1029·0 at 50° F.:

Name.	Age.	Occupation.	Habits of Life.	Hereditary Predisposition.	General state of Health.	No. of Attacks.	Cause of.	Symptoms during Attack.	State of Blood.
								pulse 96, hard; tongue white, furred. Pitting of wrist and hand on pressure. Endocarditis (slight), old probably.	no trace of uric acid.
Elizabeth Eck.	43	Widow.	Regular.	To gout from father. Two brothers had gout, and one acute rheumatism.	Very weakly.	First attack.	None discoverable.	Commenced in left hip, then in ankle and knee; after wrists and dorsum of hands. Pitting on pressure. Pulse 108, full and hard; tongue furred; thirst; no appetite. No heart affection.	Clot firm, large, buffed, slightly cupped; serum alkaline; sp. gravity 1026·4 at 55° F. No trace of uric acid.
Eliz. Fisher.	18	Lives at home.	Regular.	To rheumatism from mother.	Not strong.	Fifth attack. First at 9 years of age.	Cold.	Commenced in ankles, afterwards in knees, shoulders, elbows and wrists, and joints of hands. Pulse 135, hard; tongue furred; perspiration moderate; skin hot. No heart affection.	Clot buffed, cupped; serum alkaline; sp. gravity 1028·0 at 63° F. No trace of uric acid.
Jane Shay.	38	Married.	Not stated. Lives poorly, insufficient food.	No tendency to gout. Father has rheumatism.	Good.	First attack.	Damp residence; insufficient clothing. Nursing child when admitted.	Commenced in palmar surface of limbs, great toe much affected; also dorsum of foot; then hip, right arm, wrist, hand, &c. No heart affection. Pulse 100, hard; tongue furred; thirst.	Clot firm; serum alkaline; sp. gr. 1028·8 at 52° F. No trace of uric acid.
Amel. Ranson.	25	Servant.	Regular.	None stated.	Good, except rheumatism.	Second attack. First attack 9 years since;	—	Commenced with erythema nodosum, then joint affection; both hands, elbows, knees, and feet. No	Clot slightly buffed; serum alkaline; specific gravity 1030·8 at 57° F.

						all joints affected.		heart affection. Pulse 108; tongue furred; thirst, &c.	No trace of uric acid.
Eliza Wynne.	34	Married.	Regular.	Not stated.	Good until lately; since last confinement has felt very weak.	First attack.	Nursing at the time. No very evident exposure.	Commenced in joints of lower extremities; then shoulders, elbows, wrists. No cardiac affection. Pulse 92; tongue furred; thirst.	Clot buffed, much cupped; serum alkaline; sp. gravity 1030·0 at 50° F. No uric acid.
M. A. Sheen.	22	Servant.	Lived pretty well; worked hard in general.	None to gout or rheumatism.	Good.	First attack.	None known.	Commenced in right wrist, then right shoulder. Slight endocarditis. Pulse 116; thirst; perspiration.	Clot buffed, contracted and cupped; serum alkaline; sp. gr. 1030·4 at 53° F. No uric acid.
Mary Barry.	16	Servant.	Regular.	None to gout or rheumatism.	Good.	First attack.	Not known. Lived in London a month.	Commenced in left wrist, then right; then right foot. Pulse 112, rather hard; tongue furred; no marked perspiration. Some pitting over dorsum of foot. No cardiac affection.	Clot buffed, moderately cupped; serum alkaline; sp. gr. 1028·4 at 68° F. No trace of uric acid.

Name.	Sex.	Account of Patient.	State of Blood.
N. N—.	M.	Acute rheumatism, diagnosed by a physician.	Clot buffed; serum alkaline; sp. gr. 1030·0 at 56° F. Uric acid thread experiments gave no trace of uric acid.
M. C—.	M.	Patient suffering from sub-acute rheumatism. Commenced about 1½ year since in wrist and ankle, then other joints. Great toe never affected. No gout in family.	*Blister serum* alkaline; sp. gr. 1024·0 at 66° F. No trace of uric acid by uric acid thread experiments.
A. K—.	M.	Acute rheumatism, diagnosed by a physician.	Clot firm, buffed, and somewhat cupped; serum alkaline; sp. gr. 1028·0 at 60° F. Uric acid thread experiments gave no trace of uric acid.
A. B—.	—	Patient with acute rheumatism and pericarditis.	*Blister fluid;* serum alkaline; sp. gr. 1026·64 at 60° F. No trace of uric acid by uric acid thread experiment.
G. C—.	M.	Sub-acute rheumatism of several years' standing. Great toe never affected; weather, and not diet, produces exacerbations.	*Blister serum;* serum alkaline; sp. gr. 1023·0 at 60° F. No uric acid by uric acid thread experiment.

TABLE III.—*Articular Affection connected with Urethral Inflammation.*

Name.	Sex.	Account of Patient.	State of Blood.
F. Neville.	M.	Joint affection, following urethral affection, and afterwards accompanied by it. Much febrile disturbance, and several joints affected (knees and ankles), iritis afterwards. No heart affection.	Clot buffed and cupped; serum alkaline; sp. gr. 1028·2 at 65° F. No trace of uric acid by uric acid thread experiment.
John Connell.	M.	Joint affection, chronic, following urethral discharge. No cardiac affection.	Clot firm, buffed; serum alkaline; sp. gr. 1024·68 at 74° F. No trace of uric acid by uric acid thread experiment.
Jos. Diplock.	M.	Joint affection, connected with urethral affection. No cardial affection.	Clot normal; serum alkaline; sp. gr. 1027·2 at 66° F. No trace of uric acid by uric acid thread experiment.
— Grant.	M.	Joint affection connected with urethral discharge, large joints. No cardiac affection.	Clot normal; serum milky; sp. gr. 1026·8 at 52° F. No uric acid by uric acid thread experiment. *Blister serum;* sp. gr. 1022·8 at 52° F. No uric acid by uric acid thread experiment.
— Thomas.	M.	Chronic rheumatic affection, possibly syphilitic. Large joints affected. No cardiac affection.	Clot slightly buffed and cupped; serum alkaline; sp. gr. 1028·0 at 65° F. No uric acid by uric acid thread experiment.
Wm. Grand.	M.	Rheumatic or neuralgic affection of many of the larger joints. No cardiac affection.	Clot slightly buffed and cupped; serum alkaline; sp. gr. 1031·0 at 65° F. No trace of uric acid by uric acid thread experiment.

TABLE IV.—*Non-articular Affections.*

No	Sex.	Disease.	Uric Acid.		No	Sex.	Disease.	Uric acid.	
1	M.	Albuminuria	Blood serum gave no indication		31	M.	Accident, surgical case	Blood serum gave no indication	
2	M.	,,	,,	,,	32	F.	Surgical patient (Bursitis)	,,	,,
3	M.	,,	,,	,,	33	F.	Quartan ague	,,	,,
4	F.	,,	,,	,,	34	M.	Jaundice	,,	,,
5	M.	,,	,,	a moderate amount	35	M.	Conjestive headache	Blister serum	,,
6	M.	,,	,,	a few crystals	36	F.	,,	Blood serum	,,
7	F.	,,	,,	a moderate amount	37	M.	Paraplegia	,,	,,
8	F.	,,	,,	,,	38	M.	,,	,,	,,
9	M.	Albuminuria, 1st blood	,,	considerable ,,	39	F.	Hemiplegia	,,	,,
		— after recovery	,,	scarcely a trace	40	M.	Epilepsy	,,	,,
10	F.	Acute eczema	,,	no indication	41	M.	,,	,,	,,
11	F.	Eczema	,,	,,	42	M.	,,	,,	,,
12	M.	Eczema impetigenosis	,,	,,	43	M.	Wrist drop	,,	,,
13	F.	Psoriasis	,,	,,	44	M.	Colic	,,	,,
14	M.	,,	,,	,,	45	F.	Peritonitis	,,	,,
15	F.	Lepra	,,	,,	46	M.	Ascites	Blister serum	,,
16	F.	Acute pleurisy	,,	,,	47	M.	Anasarca	,,	,,
17	M.	Pleurisy with effusion	Blister serum	,,	48	M.	Renal calculus	Blood serum	,,
18	F.	,,	Blood serum	,,	49	M.	Suspected calculus	,,	,,
19	M.	Chronic pleurisy	Blister serum	,,	50	F.	Diabetes mellitus	,,	,,
20	M.	Pleuro-pneumonia (acute)	Blood serum	,,	51	M.	After fever	Blister serum	,,
21	F.	Pneumonia	,,	,,	52	M.	Acute inflam. of the eye	Blood serum	,,
22	M.	,,	,,	a few crystals	53	F.	Inflammation of eye	,,	,,
23	M.	,,	,,	no uric acid	54	M.	,,	,,	a few crystals
24	F.	Bronchitis	,,	no indication	55	M.	Hypertrophy of liver	,,	no indication
25	M.	,,	,,	considerable amount	56	M.	Disease of spleen	,,	,,
26	M.	Phthisis	Blister serum	no indication	57	M.	x. x.	,,	much uric acid
27	M.	Phthisis, 1st stage	,,	,,	58	M.	x. x.	,,	,,
28	F.	Phthisis and bronchitis	,,	,,	59	M.	x. x.	,,	,,
29	F.	Phthisis	,,	,,	60	M.	x. x.	,,	,,
30	M.	Emphysema	,,	,,	61	M.	Collapse of cholera	,,	,,

Table I contains a more or less detailed account of 47 patients suffering from articular disease, in whom the examination of the blood was made, and it will be found that there are many peculiarities both in the history of such patients, and in the symptoms exhibited by them; the most important are as follow:

Sex.—Only two patients out of forty-seven affected with articular disease, whose blood contained an abnormal amount of uric acid, were females.

Age.—The average of the ages of those patients in whom this point was noted, amounting to thirty-two in number, was forty-seven years; this applies to the ages of the patients when in the hospital, and not when first attacked with the disease.

Hereditary predisposition.—In twenty-six cases, it was found that thirteen, or one half, could trace what they called gout to some close blood relation. In patients in hospitals, it is not unfrequently difficult to get very clear accounts of family peculiarities, or hereditary predispositions; in the remainder of the cases, no account of such matters had been noticed.

Habits, &c.—Out of twenty-eight cases where the habits of the patient were particularly given, twenty-one acknowledged themselves to be free livers, and for the most part took largely of malt liquors, either with or without spirits, not uncommonly combined. Seven said they were very temperate, but most of these, which included the two female cases, strongly inherited the articular affection.

Occupation.—The occupation was noted in thirty-three cases; and it is very singular to observe, that eight of these or nearly twenty-five per cent., were painters or plumbers, at any rate had made use of lead in their work, and had been affected with lead disease. Any comment on this point, however, we must defer to a subsequent paper. The occupations of the remaining patients were very miscellaneous:—brewers' men and wine-coopers, gentlemen's servants, cabmen, &c. No one occupation, with the above exception, appeared to have much influence; but in those

in which the patients could readily procure malt liquors, the cases were the most numerous; but this refers more especially to the habits of the individuals.

Exciting Causes of Attack.—The causes to which the patients especially referred the attacks under which they were suffering at the time, were noted in eighteen cases. Many could assign no cause for any given attack. Of these eighteen patients, seven referred it to over-drinking, two to drink combined with fatigue or debility, two to cold, one to cold and debility, two to severe injury; in two, it came on during the course of some chest affection, and in two other cases, after epilepsy. So that over-drinking seems to be by far the most powerful exciting cause; and it is not every patient who will readily acknowledge to this fault, or it is most probable the proportion would have been much greater.

Symptoms, Affection of ball of Great Toe.—In thirty-five cases, the fact as to whether the patient had suffered from any special affection of the ball of the great toe, either in the attack under consideration or any prior one, was particularly dwelt upon; and it was found that, in twenty-nine cases, the great toe affection had been well marked, in two it was absent; in the remaining four, the feet only were affected, and in some of these latter the presence or absence of the special affection of the toe could not be positively asserted. It will be observed that in the first attack this part was more *especially selected* by the disease.

Œdema of affected parts.—Œdema occurring on the subsidence of the inflammation of the affected parts, especially the dorsum of the hands and feet, or where the surface was much affected, was noticed in twenty-four cases to be present. It was never *stated* to be absent; in many cases, desquamation of cuticle was found—a symptom doubtless intimately connected with the œdema.

Concretions or Tophaceous Deposits, or Chalk Stones.—In thirty-seven cases, the presence or absence of concretions of urate of soda upon the surface, or in such situations as to be undoubtedly recognised, was noted, and they were found

to be present in seventeen patients—absent in twenty. Some points of interest with regard to their locality were also observed. In the above seventeen cases, they occurred in the ears (on the surface of the cartilages) alone, in seven cases; in ears and around joints, &c., in nine; and in one case only could they be recognised in the other parts of the body, without also being present in the ears. Of this peculiar selection of the ear for the deposition of urate of soda, I have seen many examples in private patients, of which I have notes; and I may state, that they fully agree with the results contained in the table. These deposits may vary very greatly both in number and size, from one to eight or ten, and from the size of a small pin's head to a pea; they are beautifully crystalline, and of a consistence varying with the time at which the matter was thrown out. A representation of an ear, pretty freely studded with deposits of urate of soda, will be seen in the annexed drawing; it was made from a patient in No. 1 Table, whose blood was examined, and found to be rich in uric acid.

Acute Heart Affection, Peri- or Endocarditis.—In no one case was recent endo- or pericarditis found. Some had slight old valvular disease.

From a review of the symptoms exhibited by patients in Table I, it is evident that the majority of them are such as no physician would hesitate to affirm to be those of *true gout*, and in some, whose symptoms were not so striking during the attack under consideration, the history at once gives the clue to the nature of the disease: still there are a few, where no hereditary predisposition could be discovered, who never had the great toe specially affected, who never appeared, from their own statements, to have lived very freely, whose symptoms might, according to the definition of the diseases gout and rheumatism usually given, be referred either to gout or rheumatism, provided that the condition of the blood, or the effects produced upon the disease by remedies, were not taken into consideration; and it is the true nature of such cases that it is the especial object of the present communication to endeavour to elucidate.

Very many patients called the disease under which they were labouring, *rheumatic gout*, and on questioning them, said that their former medical attendants had so called it: as a rule, however, it was not the really difficult cases which were so named, but those in which the patient had formerly suffered from acute gout, but which disease had, in process of time, merged into a chronic affection. Not unfrequently, these so-called rheumatic gout patients exhibited abundance of chalk-like deposits of urate of soda in different parts of the body. With regard to the amount of uric acid contained in the blood, I think that it bears no direct proportion to the intensity of the local symptoms; often, I believe, an inverse ratio may hold good, as I have reason for suspecting that the local inflammations tend greatly to destroy this body, and therefore, in cases where the joint affection has remained a long time, we should not be surprised to find it greatly diminished. This was evidently the case in the blood taken from the patients Rumsey, Heath, and Rous, in Table I. At first, in Heath's blood it was not detected, but this, probably, was from the too rapid drying of the serum; it being in summer, and I not then taking all the precautions enjoined in my former communication this session. I hope to recur to this subject at a future time, but I would advise, that in cases of this kind, the serum should be put up in rather large quantities, as from a fluid drachm and half, to two drachms, and allowed to dry carefully and slowly. The presence of other inflammatory disease will probably also tend to lessen the amount of uric acid in the blood, as appeared to be shown in Johnston's case: here, however, the joint affection was by no means of a severe character.

Table II, contains an account of thirty-five cases of articular disease, not connected with urethral affection, and in which no uric acid was found in the blood. On making an analysis of these cases similar to the last, the following facts are eliminated.

Sex.—Out of the thirty-five patients, twenty-one were females, and fourteen males.

Age.—In thirty cases the age was determined, and the average was thirty years; as in Table I, the ages were those of the patients at the time of the attacks for which they were admitted.

Hereditary Predisposition.—In twenty-four cases where an account was obtained as to the existence or non-existence of hereditary predisposition to the disease under which the patients were suffering, it was found, that its existence could be made out in eight cases, its non-existence in fourteen, in two there was considerable doubt.

Habits of Patients.—Of twenty-seven cases, twenty-six considered their mode of life very regular, and that they took no unusual amount of malt liquors, wine, or spirits. In one case the patient had lived freely, and taken a considerable amount of alcoholic fluid.

Occupation.—No peculiarity was observed on this point in any of these patients.

Exciting Causes of Attack.—In eighteen patients, some cause of the attack was ascertained: of these, sixteen referred it to direct exposure to cold, and two to debility. In no case could excess of drink be ascertained to be the exciting cause.

Symptoms, Affection of Great Toe.—In thirty-one cases, where the symptoms of the present and prior attacks were given, thirty had never experienced any great toe affection, in one patient only was it stated to have occurred, and then the plantar surface of foot was also much affected, and during the attack, which was the first, the joints of the upper extremities were inflamed equally with those of the lower.

Œdema of the inflamed parts.—The presence or absence of this symptom was noticed in thirty-one cases. In twenty-seven it was absent, and in four only present to a marked degree.

Concretions of Urate of Soda or Chalk Stones.—Not present in any case examined, though particularly looked for in those in which the symptoms are detailed.

Acute Heart Affection, Peri- or Endocarditis.—In thirty-one cases examined, recent peri- or endocarditis was present in thirteen, and absent in eighteen patients.

As will at once be seen, the majority of these cases were such as physicians would pronounce to be true rheumatism, simply from a consideration of the symptoms: in some, the histories would readily clear up the diagnosis, but, as in Table I, a few remain, where the condition of the blood must be looked for to enable us clearly to refer them either to gout or rheumatism.

The most important results obtained from Tables I and II may be conveniently summed up as follows. In

Articular Affection with Uric Acid Blood.

The average age of patients was	47 years.
The males formed	about 95· per cent.
Hereditary predisposition was traced	in 50·0 „
Free living and drinking had existed	75·0 „
Painters or plumbers formed	24·3 „
Drink acted as the exciting cause	in 39·5 „
The great toe had been specially affected	82·9 „
No great toe affection	5·7 „
Doubtful	11·4 „
Œdema noticed	68·5 „
Deposits of urate of soda	45·9 „
Acute cardiac affection	none.

In Articular Affections (Non-urethral) with the Absence of Uric Acid in the Blood.

The average age	was 30 years.
The males formed	but 40·0 per cent.
Hereditary affection was traced	in 33·0 „
Cold acted as an exciting cause[1]	88·8 „
Œdema noticed	12·9 „
Acute cardiac affection	41·9 „
Deposits of urates of soda	none.
Great toe *especially* affected in	none.

[1] And alcoholic fluid did not appear to be either a predisposing or exciting cause.

In Table III will be found the results of the examination of the blood in 6 patients; in whom, although the joint affection simulated very closely true rheumatic disease, yet were separated from the cases in Table II on account of a clear relation being established with urethral inflammation; it was not thought necessary to enter into detail with regard to these; suffice it to say, that the larger joints were generally most affected, that in none was cardiac affection present, and that the febrile disturbance was by no means proportionate to the joint affection, when compared with genuine acute rheumatism, thus separating them from the cases in Table II; and from those in Table I, the want of special great toe affection, and the absence of uric acid in the blood, at once serves to remove them completely.

All the patients in this Table were males.

Table IV gives the results of the examination of the blood from sixty patients suffering from various diseases, and it will be noticed that uric acid was stated to be absent in forty-seven, and present in thirteen. On making an analysis of these thirteen cases, it is found that five were patients suffering from albuminuria, temporary or permanent, a disease which, as I have observed in a former paper, may or may not be accompanied with excess of uric acid in the blood; and the above results fully confirm my former statement, for we also find in Table IV other cases of the same disease where no uric acid was discovered. One was a case of cholera, and during collapse both urea and uric acid are retained in the circulating fluid (I might have given other analyses in this disease, showing this fact, but they have already been brought forward in a paper on the 'Pathological Condition of the Blood in Cholera.') In a specimen of blood from a surgical ward, stated to be from a patient with inflammation of the eye, a trace of uric acid was exhibited by the thread experiment; nothing was known of the case, whether gouty or not: again, a few crystals were seen in a case of a man with pneumonia, and much in one with bronchitis,—with regard to the pneumonic pa-

tient, it could not be discovered that he had ever had gout, and a very small amount only of the acid was found; the bronchitic man, although nothing is stated in his history as to any hereditary predisposition to gout, yet exhibited peculiar nervous symptoms not at all unlike those which precede a gouty attack, and these perhaps may be explained by the condition of the blood. In Table I is contained a case which bears upon this subject; the patient, Clubb, was admitted for chest affection, bronchitis and emphysema; the blood was examined, and found loaded with uric acid; the affection did not yield to the ordinary treatment for bronchitis, but after a few days the chest symptoms almost instantaneously vanished upon the appearance of gout in the great toe and knee.

With regard to the remaining four cases, marked x, x, I may state, that they were not suffering from articular disease; and the nature of the affection is withheld on account of the subject having much interest, and being at present under investigation.

We must, in the examination of blood taken from various patients, expect to find now and then some uric acid, although its presence may have no connexion with the disease under which the patient is suffering; for when once the gouty diathesis is established, the blood, even in the intervals of the attacks, seldom becomes pure: this remark applies, no doubt, with much greater force to cases in which tophaceous deposits have taken place, and perhaps more to the asthenic than sthenic forms of the affection; for it is in such cases, more particularly, that the amount of uric acid eliminated by the kidneys is found so greatly below the normal average. It will be observed, that all the patients suffering from non-articular disease, and in whose blood uric acid was found, were males; this point is interesting, when connected with the fact of the much greater frequency of gout in the male than in the female sex. I have, however, occasionally seen the most severe forms of gout, with excessive chalky deposits, in the female.

In conclusion, as we have found that the blood in every

patient suffering from genuine gout, contained an abnormal amount of uric acid, and that in acute rheumatism such was not the condition of this fluid; and again, that in all cases which could be traced up to gout (although the symptoms exhibited at the time might not be very characteristic), uric acid was present, whereas it was absent in those cases where no such phenomena could be found, I think we shall in future be fully justified in considering this condition of the blood as not only a most important, but even a *pathognomonic* sign, and one more to be depended on than any of the other symptoms taken separately; and that in an otherwise doubtful case, where the diagnosis rests between gout and rheumatism, the presence or absence of this acid in the circulating fluid (determined either from the examination of the serum of the blood or blister exudation) may be looked upon as decisive of the question. I have little doubt, but that many of the cases of rheumatism which have been described by different authors, especially the capsular form of Dr. Macleod, are really gouty in their nature. Many, however, are neither gouty nor rheumatic, and evidently closely connected either with urethral affection or purulent condition of the blood. It will be seen, that the specific gravity and reaction of the blood has been noticed in the majority of cases; with regard to the latter property I may state, that I have *always* found it *alkaline*, both in gout and rheumatism, and I am perfectly confident that the opinion which has been held by some, to the effect, that in acute rheumatism it becomes acid, is completely erroneous. On calculating the specific gravity of the serum of the different bloods, reduced to a uniform temperature, the average was found to be rather less in gout than in rheumatism, but to so small an amount that nothing valuable in diagnosis could be obtained from it. It would also be necessary, before placing any value on this fact, to eliminate from the calculation certain cases in which the specific gravity of the blood might have been altered by other causes than the diseases under consideration. With regard to the urine of gout and rheumatic patients, nothing is mentioned in the present paper; the

omission has been intentional, for I do not consider that our present knowledge of the subject is sufficient to enable us to make use of it in the diagnosis of obscure cases of these diseases. The difference between the condition of gouty and rheumatic urine becomes characteristic only when the other symptoms or signs are so. Much that is erroneous on this subject is doubtless often entertained, the appearance of copious deposits of urates being taken as indications of excess of uric acid in the blood; the converse, however, is more frequently correct, and impurity of the blood from urates is usually dependent on their deficient elimination by the kidneys.

POSTSCRIPT.—*October* 10, 1854.

Since the above paper was read, I have examined the blood in fourteen cases. In four patients there was an abnormal amount of uric acid. Three were males: all had had a special great toe affection: two had chalk stones in ears and around joints: one was a painter, and had suffered from lead colic. In five cases, where the symptoms were those of genuine acute rheumatism, no uric acid was detected: the remaining six were cases of a miscellaneous character, and the blood was free from the acid.

CASE

OF

TRAUMATIC ANEURISM

OF THE

OPHTHALMIC ARTERY,

CONSEQUENT ON INJURY OF THE HEAD,

CURED BY LIGATURE OF THE COMMON CAROTID ARTERY.

BY

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Received June 27th.—Read June 27th, 1854.

THE relation of the ophthalmic artery to the optic nerve is so close, that any serious enlargement of the vessel must be very liable to affect the function of vision, and it is therefore fortunate, that the artery is so situated, and so well protected, as to be very little subject to disease or injury. It appears, however, to have suffered in a few instances of injuries of the head, and as the changes which take place under these circumstances have not been particularly noticed, I venture to submit the following case to the consideration of the Society.

J. M., æt. 49, a labourer, was admitted into the London Hospital, March 24th, 1854, with a fracture of the clavicle, and considerable hemorrhage from the right ear, and labouring under the symptoms of concussion. He was by no means a robust man, and his hands were contracted and crippled by rheumatism. It appeared, that he had fallen from the top of a stack of wood, seven feet in height, and pitched on his right shoulder and right side of the head.

He was stunned by the fall, and remained quite unconscious until after his admission into the hospital. There were only slight marks of contusion on the head, and no evidence of fracture of the skull. Shortly after being placed in bed he vomited, and he remained in a semi-conscious condition, with a feeble pulse, for several hours. The head was shaved, a cold lotion kept to it, and a smart purge given. On the following morning he was more conscious, and after remaining for three days very restless, he seemed to be improving. The hemorrhage from the ear was followed by a serous discharge for about a week, and by total deafness of the right ear, and he complained of a dull aching pain on the right side of the head. In about a week after the accident, the face was observed to be drawn slightly to the right side, but the tongue could be protruded straight out of the mouth. He was blistered behind the right ear, and ordered to take small doses of calomel; and as he was in a weak condition, his diet was improved. In a fortnight later, the paralysis of the right side of the face had nearly subsided, and the pain in the head had diminished. About the beginning of May, I noticed a little inflammation of the conjunctiva of the right eye, attended with slight chemosis. For this, a lotion consisting of a weak solution of the nitrate of silver, was ordered. The injection of the conjunctiva and chemosis continued, however, to increase, and the eye-ball was observed to be prominent. I then suspected that some mischief was going on at the bottom of the orbit. There was also more pain in the head. An issue was made behind the ear, and the nitrate of silver lotion to the eye was discontinued. Fomentations were substituted, and two leeches were applied to the right temple, and repeated two or three times. This relieved the pain in the head, but had no effect on the eye.

May 22d.—The eye-ball protruded so much, that I was induced to make a careful examination of the orbit, which led me to detect a pulsation on placing the finger on the upper lid, and pressing gently on the globe. The proptosis

was more marked two days afterwards, and a very distinct *bruit* was heard when the ear was placed against the patient's right temple. He also described the pain in the head as a distressing throbbing sensation. Vision was not all impaired; but he had very little power of moving the eye. He was kept at rest in bed, with the head elevated, and a small bladder of ice was applied over the orbit; but this was so uncomfortable, that it was discontinued after two days.

31st.—A consultation was held to consider the propriety of tying the carotid artery. The patient had lost the left eye from cataract ten years before, which rendered it of greater consequence to save the right. The sight of this eye was somewhat impaired, but as he could see, with a little difficulty, to read small letters, and vision was not decidedly injured, and the proptosis had not perceptibly increased for two days, it was decided to wait.

June 2d.—Finding the eye getting more prominent, and vision becoming impaired, the pupil being widely dilated, I determined on tying the right common carotid artery. The ligature was applied on the vessel in the upper third of its course. The pulsation of the eye-ball was at once arrested, and the man was relieved of the beating pain in his head. A dose of morphia was given at bed-time.

3d.—The man had slept only one hour. He experienced pain in swallowing, and had slight twitchings of the muscles. The eye was less prominent, but vision was not so good as before the operation.

4th.—He was unable to discern the objects before him; indeed, his vision was lost. He could only distinguish between light and darkness. The pupil was dilated, and the iris did not act on exposure of the eye to strong light. He was otherwise doing well, and felt no pain in the head.

5th.—The chemosis and redness of conjunctiva had nearly disappeared, and the projection of the eyeball had almost subsided. The cornea was dull and hazy. The grey iris was slightly discoloured, of a greenish hue, and the pupil was widely dilated, of an irregular oval form from old adhesions. He experienced considerable intolerance of light.

On the 8th the cornea was observed to be less hazy, and by the 11th had become nearly clear. Vision was returning, but the intolerance of light continued. He had quite recovered the power of moving the eyeball, which had subsided to its proper place in the orbit. He could hear better on the right side, and the facial paralysis was scarcely perceptible. He eat and slept well. On the 13th he could distinguish objects held before him. The pupil, however, remained dilated, and the iris motionless under the stimulus of light. In about a week later the intolerance of light passed off, and he was able to discern objects at a distance pretty clearly, but not near objects. He could not see to read, nor make out the hands and figures of a watch. On looking, however, through a small hole in a card, he was able to see much better, and could read with a little difficulty, and ascertain the time on the face of a watch. The ligature came away on the 23d day. P.S.—The patient was discharged from the hospital at the end of July. His vision was at that time much improved. The pupil was less dilated, but still fixed.

The history of the above case clearly shows, that a severe injury of the head had been the occasion of the formation of an aneurism of the ophthalmic artery. The bleeding from the ear, the subsequent discharge of serum, and total deafness of the right ear, and paralysis of the parts supplied by the portio aura, indicated some serious injury to the base of the skull, and it seems probable that the petrous portion of the temporal bone was fractured, and that by the extension of the fracture to the optic foramen the ophthalmic artery had been wounded by a splinter or detached fragment of bone. There was no indication, however, of an aneurism having formed until upwards of five weeks after the accident. Its progress was then slow, and vision was not affected until nearly a month after the first appearance of anything wrong in the orbit, and was only slightly impaired before the operation.

I was unwilling to place a ligature upon the carotid, which in a person of feeble health and weak power was not unlikely

to produce cerebral mischief, without an urgent necessity for the operation; but the previous loss of the left eye rendered the preservation of the right of greater consequence. I watched the case, therefore, with anxiety from day to day, having resolved to tie the carotid artery immediately that vision was seriously threatened; and having performed the operation, apparently in good time to save the eye, I was greatly mortified to find sight entirely gone on the second day afterwards. This occurrence, and the recovery of sight in a short time, are circumstances of much interest in the history of the case. The temporary loss of vision must be ascribed to changes consequent on defective nutrition, from the arrest of the circulation through the carotid artery, the aneurismal tumour interfering probably with the supply of blood to the eyeball from collateral sources. But as the proptosis subsided, and the circulation became reestablished, the eye recovered its nutrition, the cornea became transparent, and sight returned. The remarkable dilatation of the pupil, which continued after the recovery of vision, cannot, I think, be referred to the same cause. It seems most likely to be due to the aneurism pressing on or stretching the ciliary nerves, and destroying their functions, as respects the motions of the iris, producing, in fact, mydriasis.

This case clearly establishes the great danger to vision caused by a traumatic aneurism of the ophthalmic artery, from pressure on or traction of the optic nerve, and the ciliary nerves. It also shows, that to avoid these sources of danger, as well as to prevent the risk of the eye being injured by impeded nutrition after operation, a ligature should be applied to the common carotid artery at an early period, or soon after the detection of the pulsating projection of the eye-ball.

In 1834, I witnessed, at the London Hospital, a case of a similar nature to the one just related. A youth sustained a fall which produced concussion, attended with proptosis, dilated pupil, and loss of vision of the right eye. The prominence of the eye-ball increased, and at the end of a

month pulsation was detected. During a fit of coughing, violent arterial hemorrhage occurred from the nose, when Mr. Scott, who was at hand, instantly tied the right common carotid artery. The proptosis subsided, but vision remained permanently lost. Mr. Busk, in a brief notice of this case, justly remarks, "the protrusion of the globe immediately after the accident, without symptoms of cerebral compression, proved that it arose from extravasation of blood within the orbit, and the further continued protrusion rendered it probable that the aperture in the vessel from which the blood escaped had not closed.

These two cases, and the interesting one related by Mr. Busk, in the 'Society's Transactions' (vol. 22), are, I believe, the only examples of aneurism of the ophthalmic artery, consequent on an injury of the head, on record. In Mr. Busk's case, it appears that a seaman was rendered insensible by a severe blow, which was followed by bleeding from the right ear and deafness, with paralysis of the left side of the face, and immobility of the left eye, with dilated pupil. Suppuration of the cornea ensued, and ended in an opaque cicatrix of its lower half. About seven months after the accident, Mr. Busk detected a small pulsating tumour in the left orbit, and tied the left carotid artery. The patient recovered, with vision through the upper part of the cornea, but with a fixed pupil. The proptosis appears to have been but slight, and after the discovery of pulsation, the carotid artery was tied without delay.

ENGLISH STATISTICS

OF

HOOPING-COUGH.

BY

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COMMUNICATED BY

H. BENCE JONES, M.D.

Received June 26th.—Read June 27th, 1854.

Etiology of Hooping-Cough.

IN 1851, I published a series of papers in the 'Medical Times,' on the Etiology of Phthisis, chiefly prepared from returns which had been made to the General Register Office, but which have not been issued. I purpose now to solicit attention to similar statistical details in reference to another and less important pectoral affection, with a view to a more extended analysis than can be inserted in systematic works written upon that disease. I am not ignorant of such valuable summaries as have been given by Dr. West in his very excellent work on 'Diseases of Children,' and more recently (since this paper was written) by my talented friend Dr. Gibb; neither am I prepared to affirm that professional opinion on this subject is in any way inaccurate; but since much that is mysterious still clings around the origin of hooping-cough, and since the disease is, at the present time, very fatal, it seems not inopportune to offer such further observations as the present state of science will permit. As accuracy should be an essential element in all statistics, and as mere numbers of cases, without any datum from which

to calculate their true value, is of little or no avail, I shall pursue my former course, and restrict my investigations to the returns published by the Registrar-General. I do not refer to the results of personal experience, or even to those of any public institution, no matter how large either may be, since, if we were prepared to grant that such returns could be as accurate as those obtained by an institution exclusively devoted to mortality statistics, they must be on a most contracted scale, and lack that great basis of comparison—the proportion to the population.

As a preliminary remark, I may observe that, whilst this age is remarkable for the cultivation of medical statistics, we are still, *de facto,* restricted to a consideration of fatal cases only, and therefore remain wholly ignorant as to the precise prevalence of any affection. This observation, it is true, varies in value according to the nature of the disease under review; for if that disease be necessarily fatal, a knowledge of its mortality will give its real prevalence; but, on the other hand, if the given disease be but rarely mortal, so in the like proportion will the results of the mortality tables be of little avail. This is pre-eminently the case in the disease now under consideration; for if we may rely in any degree upon the results of personal experience, the fatal bear scarcely any proportion to the recovered cases; and, further, when death occurs, it is due rather to the complications of the disease than to the disease itself. Again, mortality tables afford no information upon many associated points of inquiry—as, for example, the duration of the disease, and the nature and frequency of its complications. The influence of age, sex, and season is, for the same reason, but imperfectly shadowed forth; for if we aver otherwise, we must assume that, whatever may be the proportions which these influences bear in mortal cases, they necessarily exercise the same, in the same ratio, in recovered cases. This cannot but be unsatisfactory to every inquirer, and should excite the profession, as a body, to seek a removal of the evil; but until an institution is established for the collection of vital statistics on similar

principles to those of the General Register Office, in reference to mortality statistics, or until some combined and intelligent efforts are put forth by all our medical bodies, for the like purpose, the efforts of individuals will be made in vain.

The following summary of our available information is true of hooping-cough, and almost equally true of every other disease, viz., that the frequency of its attack, and the period of its duration, is really unknown; and that the influence of age, sex, and season, is uncertainly indicated; whilst the number of the fatal cases of the disease and its complications, combined with the age, sex, and the season at and in which the deaths occurred is known with almost sufficient certainty. I shall, therefore, limit this communication to a consideration of the mortality statistics of Pertussis.

Frequency.

As the disease is for the most part not fatal, one is oftentimes struck with the reports of deaths returned in the London district, as from 60 to 80 per week in the winter and spring months, and so many as 36 per week throughout the year, on an average of 10 years past. The true importance of this amount of mortality is not perceived by the mere repetition of the numbers, but rather when it is contrasted with the mortality from some other diseases. Thus, in the same registration district, during the 10 years from 1844 to 1853, both inclusive, of the 99 diseases which the Registrar-General has selected, under which to arrange the general mortality, hooping-cough occupies no lower a rank than the 7th place from the highest. The only affections of the chest (a class of affections with which it may be associated) which have a higher mortality, are phthisis, pneumonia, and bronchitis, in their order; of members of the zymotic class (with which it is also connected), only typhus and scarlatina exceed it; and lastly, of diseases of the nervous system (with which it again has a correspondence), convulsions alone have a higher mortality.

Thus, of the 99 diseases, or classes of diseases referred to, the following alone have a higher mortality, viz., phthisis, pneumonia, bronchitis, typhus, convulsions, and scarlatina, in their order. It is a fact worthy of prominence, that the mortality from disease of the heart, hydrocephalus, apoplexy, measles, and smallpox, each in its order, is less than that of hooping-cough. This is not in accordance with popular belief, whether in its relation to some of the affections referred to, or to others which are known to be deadly, but which, nevertheless, have so slight a mortality as to rank only after the last in the list just mentioned.

Table I.

Mortality from selected diseases in the London district during the ten years from 1844 to 1853, both inclusive, arranged in the order of prevalence.

Phthisis	68·204
Pneumonia	36·494
Bronchitis	32·146
Typhus	23·107
Convulsions	21·531
Scarlatina	20·444
HOOPING-COUGH	18·666
Disease of the Heart	17·647
Hydrocephalus	15·977
Apoplexy	12·629
Measles	11·627
Smallpox	9·007
Total from all causes	553·694

The like relative importance of hooping-cough is observed when the state of England and Wales is examined; for if we take the last returns published, viz., those of 1847, we find that only one additional disease takes precedence of it, viz., diarrhœa, and then the more usual diseases are phthisis, typhus, convulsions, pneumonia, bronchitis, scarlatina, and diarrhœa, in their order. There is, however, some discrepancy when we analyse the great divisions into which

England is divided, for there we discover that in the eastern and York divisions, there are only three or four more fatal diseases; whilst on the other hand, in the south-western, there are no less than sixteen diseases which to a greater or less extent take precedence. As it may be of interest to notice the diversity in the relative mortality from various diseases in the great divisions of England, I have prepared the following Table:

Table II.

The Diseases in their order of mortality which were more mortal than Hooping-Cough in 1847 *throughout the Registration Divisions (exclusive of London) in England.*

South Eastern. No. 2.	South Midland. No. 3.	Eastern. No. 4.	South Western. No. 5.	West Midland. No. 6.	North Midland. No. 7.	North Western. No. 8.	York. No. 9.	Northern. No. 10.	Welsh. No. 11.
Phthisis 4507	Phthisis 4569	Phthisis 3457	Phthisis 4743	Phthisis 5976	Phthisis 3505	Typhus 9076	Phthisis 5228	Phthisis 2649	Phthisis 3629
Typhus 1884	Typhus 2030	Pneum. 1286	Pneum. 2230	Typhus 3388	Convul. 2187	Phthisis 9044	Convul. 3896	Scarlati. 2012	Convul. 2524
Pneum. 1831	Pneum. 1529	Typhus 1262	Typhus 1662	Pneum. 3307	Typhus 1664	Convul. 5399	Typhus 2900	Typhus 1304	Typhus 1966
Convul. 1442	Convul. 1027		Dropsy 1634	Convul. 2506	Pneum. 1428	Diarrh. 3868	Scarlati. 2279	Pneum. 973	Smallpx 1003
Bronch. 1300	Dropsy 919		Bronch. 1212	Scarlati. 2493	Dropsy 798	Pneum. 3464	Pneum. 2221	Convul. 959	Pneum. 888
Dropsy 1161	Bronch. 779		Convul. 1109	Bronch. 1929	Bronch. 654	Scarlati. 3439		Dropsy 718	Dropsy 868
	Apoplexy 597		Apoplexy 946	Dropsy 1608	Measles 645	Bronch. 2979		Bronch. 673	Bronch. 672
	Heart 553		Heart 919	Diarrh. 1480	Diarrh. 550	Measles 2121		Hydrocc. 526	Scarlati. 479
	Diarrh. 546		Paralysis 748	Measles 1143	Heart 510	Dysent. 1632		Diarrh. 519	Measles 547
	Scarlat. 542		Hydroce. 577	Heart 1065	Scarlati. 480			Heart 486	Asthma 455
	Paralysis 488		Diarrh. 568					Paralysis 433	Paralysis 416
			Measles 568						
			Cancer 542						
			Inflam. 488						
			Asthma 431						
			Enteritis 372						
Hoop. Cough } 1000	381	806	363	1028	476	1608	1206	427	365

It must be remembered, when studying these tables, that whilst No. I is obtained from an average of 10 years, and may therefore express the truth, No. II represents but one year, and may be, in some of its parts, only an approximation to the truth. Moreover, the numbers now referred to, demonstrate only the actual deaths which have occurred, and not the true ratio of mortality; and it must ever be borne in mind, that the absolute mortality and the ratio of mortality are not convertible terms. The ratio of mortality can only be determined by selecting some basis for a common computation, and then computing each one separately on that basis. The basis most commonly selected of late years, is that of the population at the ages at which the deaths took place. This I shall use when considering the influence of age over the mortality from hooping-cough. The older basis of computation (or that adopted before the returns of the census were so minute and accurate as at the present time) was the total mortality in given districts; but this is of but little value, for it is a matter of common belief that the relative prevalence of many diseases, as of phthisis and typhus, for example, has varied much during the last fifty years. On this basis, the mortality from hooping-cough was to the total mortality in London, during the 10 years (1844 to 1853), as 1 to 29·6. This fact may be used as an excellent illustration of the importance of preparing statistics from a series of years, rather than from any one year; and also of taking a wide area over which they may range. Thus, in 1847, the proportion in all England and Wales, was as 1 to 45·7; and in London, in that year, instead of being as 1 to 29·6, it was as 1 to 37·. Great variation was observed in the several divisions, ranging from 1 to 94·8, up to 1 to 28·1. Thus, eastern 28·1; south-eastern, 32·8; York, 35·5; London, 37·; north-western, 49·2; west midland, 49·4; northern, 53·8; south-midland, 68·4; Welsh, 72·; and south-western, 94·8.

Recurring to the London statistics for the 10 years above mentioned, we notice that the mortality varied greatly throughout the series, and was the greatest in 1853 (50 per

week), and 1849 (45 per week), whilst it was the least in 1844 (25 per week); the average of the 10 years being, as above mentioned, 36 per week. In explanation of this fact it may be stated, that the two former years were also remarkable for their general mortality, and that cholera was epidemic in 1849; for a common belief prevails to the effect, that the degree of mortality of any disease may be influenced by the healthiness or otherwise of a season regarded as a whole. That these two facts do not run parallel in this instance, may be inferred from the following facts. The general mortality was far greater, and yet the mortality from hooping-cough was much less in 1849 than in 1853. The year 1847 had even a higher ratio of mortality than 1853, and yet the weekly deaths from hooping-cough were only 30·7 in 1847 to 50 in 1853. In 1849 and 1853, the deaths from allied nervous, pectoral, and zymotic diseases, separately, were increased with the increase of the general mortality, but not by any means in the proportion in which the mortality from hooping-cough was increased; whilst on the other hand, in 1847, the mortality from hooping-cough was very greatly reduced, and yet that from influenza was increased tenfold; and from nearly all other members of the zymotic class, with pectoral diseases, was doubled. The mortality from nervous diseases was also then increased. The year 1844, which was so exceptional in reference to the diminished mortality from hooping cough, was not in like manner exceptional in relation to the general mortality; for whilst the general mortality was low, it was yet higher than other years in the series, in which hooping-cough was more fatal.

Thus it is probable, that the mortality from hooping-cough bears no exact analogy to that from all causes, nor to that from the most closely allied classes of diseases. It is needful to proceed from the examination of generals to particulars, such as age, sex, and season, in order to discover the special circumstances which exert so important an influence over this disease.

The Influence of Age.

The influence of age is well marked, and in accordance with common belief. The latest statistics, on a large scale, are those of 1849, for England and Wales, which give the mortality from each disease at various ages. Before using these returns, it is necessary to determine the ratio of mortality as estimated from the population living at the various ages during that year. We have the returns of the census for 1841, and the proper correction for the increase of population for the six years is the addition to the census of somewhat less than a $\frac{1}{13}$th part. It is true that this correction must vary with the varying increase of population during each decennial period, and also that the rate of increase on female lives, as determined by the census of 1841, viz., 1·332 per cent., has not been confirmed by the census of 1851; yet, as the last decennial retrogression has been due to temperature and pestilence, neither of which occurrences had excited any very marked disturbing influences previous to 1847, we are, I think, justified in using for that year the old rate of increase. Whatever rate, however, is agreed upon, the relative mortality of diseases in that year is equally well determined. As I adopted that correction in the paper on the 'Etiology of Phthisis,' I propose to resume it here. The ratio of mortality from hooping-cough to the whole population, *at all ages*, when calculated on the above basis, is as 1 to 1814; whilst, for comparison, that of phthisis is 1 to 324; and of pneumonia, the second disease in point of mortality, 1 to 737. The following table exhibits the absolute mortality, with its ratio to the population at various ages, as observed in England and Wales in 1847:

TABLE III.

Age.	Deaths in England and Wales 1847.	Per Centage proportion to the whole Deaths 9260.	Ratio of Mortality to the Population at various Ages.
All ages.	9260		1 in 1814
Under æt. 1 year	3746	40·4	„ 123
1 to 2	2546	27·4	„ 182
Under æt. 2 years	6292	67·8	
2 to 3	1284	13·8	„ 367
3 „ 4	720	07·7	„ 614
4 „ 5	437	04·7	„ 991
Under æt. 5 years	8733	94·1	„ 260
5 to 10	487		„ 4222
10 „ 15	20		
16 „ 20	6		
Upwards to 90	12		
Unknown	2		
	9260		

From the above table we learn, that more than two fifths of the whole deaths occurred under 1 year of age; more than two thirds under 2 years of age, and nearly the whole, that is, nineteen twentieths, under 5 years. The chief feature in reference to the true ratio of mortality is the fearful prevalence and fatality of the disease when attacking infants under 1 year of age. This is no less than 1 to 123 living, a mortality considerably greater than that of phthisis, and beyond that of any other disease except those comprehended under the general terms convulsions, pneumonia, and diarrhœa, in their order. We may safely affirm, that hooping-cough (with its complications) is the most fatal of all diseases during the first 12 months of life. From this period it progressively and gradually declines in fatality until the commencement of the 5th year, and after the 10th year is comparatively innocuous or unknown. This fact being established, it would be proper to analyse the circumstances which attend upon the earliest months of existence, with a view to isolate those which have

an especial reference to the production of this disease, but I am not aware of any data on which we can proceed, except such as exercise an influence over the general mortality at that early period, and especially such as tend to produce convulsions, or other fatal derangements of the nervous system. There can be no doubt of the fact, that the high degree of sensibility to impression which attends upon infancy, plays a most important part in this, as in other diseases, but that it does not simply act by adding intensity to the effects accruing from changes of temperature, may be inferred from the (probably) equal prevalence of the affection amongst all classes of the community; and in like manner it may be inferred, that it does not act by the ordinary zymotic influences only, since it alone, of all zymotic diseases, nestles itself habitually in the infant's cradle.

The Influence of Sex.

The influence of sex over mortality is in general rather the indirect one of the circumstances in which each sex is especially placed, than the direct one of any peculiarity of organization. (Diseases of the sexual organs are of course excluded from this statement.) But few diseases are known to have any universal preference for the female over the male sex, although most diseases have their nervous element somewhat more developed in the former than in the latter. This latter has usually been attributed to the higher sensibility or delicacy of organization which experience has ascribed to the female sex; and since we cannot doubt that this condition of system does obtain, it would not seem an unreasonable inference if we were to infer that any affection which has its essential seat in deranged nervous function, may directly prevail in the former sex. Yet, as suggested to me by my friend Dr. Sibson, such an inference is directly negatived by the palpable fact that convulsions (in the somewhat confused form recorded by the registrar-general) are not only more frequent in males at all ages, but in infants under 1 year. It is difficult to reconcile this fact with what would

otherwise appear to be a legitimate deduction; and although at all times ready to give the preference to fact over theory, we cannot but believe that some circumstances exist, as yet unknown to us, which would greatly modify the influence of the fact just mentioned. Whether this is associated at all with the distinction between mortality and prevalence to which I have before referred, I cannot tell; but it is quite within legitimate conception, that a disease may prevail in one sex, and yet be even more mortal in the other sex. In convulsions, for example, it does not follow, that because the greater mortality has been undoubtedly observed in the male sex, that therefore the true ratio of the prevalence in numbers, of the disease, attaches to that sex, for it is one thing to have a disease, and another to die from it. Moreover, as more males than females are born, it would demand more deaths of the former than of the latter, in order to make an even ratio of mortality. Further, if we may justly admit, that there is greater sensibility to impression in the female, we may with equal truth affirm, that there are greater powers of passive endurance also; and therefore it is not inconsistent to state, that females may be more liable to a disease than males, and yet that the mortality may be really greater in males.

But if this reasoning may be allowed in reference to convulsions and other nervous affections, it does not suffice to explain the indisputable fact, that hooping-cough is much more fatal in females than in males. The female system, in reference to this disease, seems to have not only a theoretical predisposition to its attack, on account of its delicacy of organization, but a predisposition to succumb under its influence; for not only are there more females than males living at every age, but the number of deaths and the true ratio of mortality are greater in the female sex. This is a most interesting fact in relation to convulsions, and other fatal nervous diseases with which hooping-cough, in its essential character, is unquestionably allied, and one which for the present appears to be inexplicable. It may be true, that hooping-cough kills by its complications, and that these com-

plications are usually inflammatory; but that does not help us, unless we could prove by statistical facts, that the female system is especially prone to the attacks, and the fatal termination of inflammation. The fact, however, remains, that hooping-cough is more mortal in the female than in the male sex; but there are no data to show, that the female system is more prone to the attacks of the disease.

The following table shows the number of deaths from hooping-cough which occurred in each sex at various ages in England and Wales during 1847, with their respective ratios of mortality to the population.

TABLE IV.

Ages.	MALES.		FEMALES.	
	Deaths in England and Wales. 1847.	Proportion to the Population. 1847.	Proportion to the Population. 1847.	Deaths in England and Wales. 1847.
All ages . . .	4126	1 in 2044	1 in 1717	5134
Under æt. 1 year	1767	„ 143	„ 119	1979
1 to 2 years	1092	„ 212	„ 159	1454
2 „ 3	567	„ 414	„ 330	717
3 „ 4	318	„ 689	„ 554	402
4 „ 5	169	„ 1297	„ 807	268
Under æt. 5 years	3913	„ 288	„ 237	4820
5 to 10	202			285
10 „ 15	4			16
15 „ 20	3			3
Upward	4			8
Unknown				2
	4126			5134

The above table proves, not only that at *every period of* life the true ratio of mortality is higher in *females than in* males, but the yet more interesting fact, that this preponderance increases as life progresses. Thus, whilst under 1 year of age, the excess in the ratio of mortality amongst females is one sixth, it is less than one third in the fifth year of existence, and was reduced to one fourth in the second year, and one fifth in the succeeding intervals. It is unsa-

tisfactory to pursue the comparison at later periods of life, on account of the smallness of the numbers to be contrasted, but so far as this is of value, it proves that this preponderance is maintained, and even increased at puberty, and for an indefinite period beyond that æra. This curious fact sustains, in a degree, the theory above mentioned, viz., the predisposition arising from organization, since we may assume that the peculiarities of the female organization are not so distinguishingly developed within the first year as in subsequent periods of life.

The Influence of Season.

We have hitherto been unsuccessful in all attempts to determine what element of the series constituting atmospheric phenomena, has had permanent influence over any disease, although, at the same time, we know well that the atmosphere, as a whole, or by some of its component parts and properties, does exert an important influence to this end. Electricity, winds, vapour, barometric pressure, and temperature, have each been investigated, and in all cases with some success; but with the exception of the latter, the published returns for series of years, either from want of uniformity in design, or occasional omissions and alterations, are not available. Temperature is therefore the only element which we shall consider apart from the others constituting the season.

The average temperature of the 10 years selected, 1844 to 1853, was 49·3°, whilst that of the two exceptional years of great mortality from hooping-cough, viz., 1849 and 1853, was 49·9° and 47·7° respectively. The year 1853 had a lower temperature than any in the series, except 1845, which was only $\frac{1}{10}$th of a degree colder. It was otherwise, however, with 1849, for six of the ten years had a lower temperature than that year. Thus, although the first mentioned year, that of 1853, was decidedly characterised by a low temperature, and experienced the greatest mortality from hooping-cough, and so far would connect cold and

hooping-cough together, it must be remarked, that the yet colder year of 1845 had only two thirds of the number of deaths from that disease; and, on the contrary, the year 1849, which had so great a mortality from hooping-cough, was a little warmer than the average of years. Considering the year as a whole, therefore, we do not trace the connexion between excess of cold and excess of mortality from hooping-cough.

In order to study the relation between temperature and this disease, we must examine, not only the exception, but the rule, and enquire what is the ordinary occurrence at the various seasons of the year. I have, therefore, examined these two points in each quarter of the seven years, 1847 to 1853, both inclusive, and have ascertained that the greatest mortality occurred five times in the 1st, and once in the 2d and 4th quarters, and that the least mortality took place five times in the 3d, and twice in the 4th quarters. This proves that the winter is the most obnoxious, and the summer the least obnoxious to this disease. But it may be objected that the division into quarters is artificial, and that as the seasons run insensibly into each other, and yet during the year exhibit two opposite characters, it is more reasonable to convert the 1st and 4th quarters, either of the same, or better still, of consecutive years, and call them winter, and the 2d and 3d quarters and call them summer. Adopting this more natural division of seasons, I have found that the greatest mortality in the 7 years, was 5 times in the winter both of the same and of consecutive years, and the least mortality 5 times in the summer. This exhibits a remarkable correspondence with the results of the computation by quarters, and clearly demonstrates the influence which temperature exerts over this disease.

The following Tables illustrate the above remarks.

Tables V and VI.

Quarterly Mortality from Hooping-Cough in London.					Half-Yearly Mortality, all in the same year.		Half-Yearly Mortality. The Winter of two consecutive Years.			Years.
	1st.	2d.	3d.	4th.	Summer.	Winter.		Summer.	Winter.	
1853	702	857	426	667	1283	1369	1853	1283	1018	1852–3
1852	539	466	244	316	710	855	1852	710	825	1851–2
1851	781	734	360	286	1094	1067	1851	1094	1205	1850–1
1850	442	406	300	424	706	866	1850	706	715	1849–50
1849	905	739	428	273	1167	978	1849	1167	1377	1848–9
1848	374	449	340	472	789	846	1848	789	800	1847–8
1847	544	392	238	426	630	970	1847			

A complete analysis of the influence of temperature demands a yet further restriction of the period over which the average shall be carried. The returns of the Registrar-General would enable us to reduce the average of the mortality to weeks, and of temperature to days, but since in but few, if any, instances do the atmospheric conditions of a week produce immediate death, it would be needless to so far limit our attention. Persons may reasonably differ in opinion as to the length of time which may usually elapse between any atmospheric change and the fatal results, and therefore, as to whether the lowest analysis should be that of 2, 3, or 4 weeks. As the selections must be arbitrary, but yet so far founded on observation, I think that monthly periods would probably exclude mere occasional influences, and connect together, by an average, the cause and its effect. I have adopted this plan, and have deduced the *weekly* average both of mortality and temperature from the totals of each concluding month in the years 1847 to 1853, both inclusive.

TABLE VII.

The mean Weekly Average of Monthly Mortality and Temperature, London.

Month.	1847.		1848.		1849.		1850.		1851.		1852.		1853.		Month.
	Mort.	Temp.	Mort.	Temp.	Mort.	Temp.	Mort.	Temp.	Mort.	Temp.	Mort.	Temp.	Mort.	Temp.	
January .	44	34·4	32·5	34·4	60	40·5	30·2	32·3	48	40·1	52	41·2	39·6	44·3	January
February .	41·5	35·4	25·5	43·2	60·5	42·9	34	43·9	54·5	41	38·5	41	58·5	33·7	February
March .	39·5	49·7	26·2	42·5	76·6	42·4	37	39·9	74·4	41·7	43·5	40·6	66	37·4	March
April . .	35·5	53·2	38	47·9	58·2	42·6	33·2	47·8	59·2	45	41·5	45·1	74·5	45	April
May . .	33	55	36	69·5	61	53·1	36	49·2	63	48·5	37·6	51·1	63	50·1	May
June . .	21	57·6	28·7	59·5	52·4	56·7	26	60·3	45·2	57·8	29	55·3	61.2	57	June
July . .	18	64·2	28·6	61·8	37·2	62·2	23·2	62	35·7	60·1	18·4	65·7	40	60·3	July
August .	17·5	62·3	22·5	57	35·2	62·2	21	61·2	28·4	62·4	16	62·2	31	60·3	August
September	19·5	55·2	27·4	57·3	27·6	57·7	25·4	56·7	18·7	55·9	22	57·9	25·5	56	September
October .	19·7	52·5	29·5	52·1	22	51·3	22·7	48·1	14·2	54	22·6	48·4	36·6	51·4	October
November	27·2	46·8	29	43·2	18·7	47·2	25·6	46	23·8	39·5	22	50·1	44·2	50	November
December	47·4	42·4	47·6	44·9	22	38·4	57·2	39·9	27·5	41	28·7	46·7	61·6	35	December

The same facts are also represented in the diagram No. I, in order that the contrast of temperature and mortality may be more quickly appreciated.

The foregoing Table and Diagram show, that the mortality and temperature are in the inverse ratio to each other, and that the former proceeds in waves from about August, when it is at its minimum, to about April, when it is at its maximum, returning to its minimum about the following August, and thus continuing in even waves of increase and decrease, with remarkable regularity, from year to year. The maximum and minimum months occasionally vary. Thus, the former, instead of being April, may be March, and in one instance, it was the preceding December; whilst the latter, instead of being August, may be July or September, and in one instance, was even November. This degree of variation, however, in no sense invalidates the rule which has just been laid down. In reference to temperature, the Diagram proves that the mark of its maximum is not that of the minimum of mortality, but the one which immediately precedes it; and so, in like manner, with the minimum of temperature and the maximum of mortality. This rule is also, like other rules, liable to exception; but when such exceptions occur, it will usually be found that the temperature, or the mortality, has remained at nearly the same point during two or three months. In such instances, it manifestly gives a false importance to one particular month over its neighbour, if it be denominated the minimum month, because it had one, two, or three degrees less temperature. Such is the relation between mortality and temperature; and after making every allowance for exceptional cases, we cannot but be struck with the regular and almost constant apposition upon the Diagram of the two lines of temperature and mortality.

The waves of the greatest intensity in the series of years referred to (1847 to 1853), occurred at intervals of two years, and were succeeded by a marked rapid and extreme subsidence in mortality during the summer and early autumn months, and did not again approach to the same

intensity during the succeeding years. Thus, in 1849, 1851, and 1853, the highest average weekly mortality in one month, was 76·6, 74·4, and 74·5, respectively, whilst in the alternate years, 1848, 1850, and 1852, the like highest average was only 47·6, 51·2, and 52·. In the latter part of the former years, however, there was not that uniformity of opposition of the lines of mortality and temperature which constitutes the rule, but, on the contrary, a disposition was manifested to pursue a parallel course. These facts may tend to prove, that a severe outbreak of the disease is followed by diminished intensity of mortality, and that, to a certain degree, in spite of the action of causes which, under other circumstances, would have heightened the mortality. This is an interesting feature, and one which it would be worth the trouble to work out through a much larger series of years, could we obtain the necessary statistical returns. It, however, probably corresponds with some other zymotic diseases. Another circumstance of interest to be gathered from the Diagram is this, that at the close of each alternate year of accession, the intensity of mortality seemed rather to move in advance than in the year of the subsidence of the temperature, in opposition to the fact just noticed in relation to the years of intensity; for, in November of the years 1848, 1850, and 1852, the mortality suddenly increased, whilst the temperature yet remained at a tolerable height, viz., 45°.

The intensity of mortality usually advances and recedes by slow and stealthy steps, but in many instances, it appears to leap suddenly in another direction, and it is not difficult to draw a line which may be considered the boundary line between low and high mortality. This lay between 45° and 48°, for it was only as the summer temperature descended to that point that the mortality assumed a decided average tendency to increase.

From all the foregoing observations, I think it is clear that mortality of hooping-cough attends diminished temperature with considerable precision, and so far may have a point of correspondence with other seasonal affections; but

there is one point in which it differs from others, viz., that it is not increased in intensity by any intensity of the opposite season, or that of summer. Excessively high temperature, so far from having given rise to increase of mortality, was directly the reverse, and when the wave of minimum mortality occurred so late as the October of 1851, it was because the average weekly temperature of that month was 54°—a temperature higher than that of the same month in any of the other years in the series.

In order the more certainly to establish the position just laid down, I considered that indirect as well as direct evidence should be adduced. I have, therefore, prepared Table IX and Diagram II, with a view to show the absolute and relative bearings of temperature with the general mortality, and the three classes of disease with which hooping-cough is associated, viz., the zymotic, the pectoral (including phthisis), and the nervous; and also with a further view of ascertaining what correspondence in these relations exists between hooping-cough, and any or all of the three classes of diseases referred to. The table has been compiled by abstracting the weekly returns of deaths from the weekly reports, and adding the numbers together by months, and then dividing the result by the number of weeks, in order to obtain the weekly average per month. The Diagram is thus one of interest from the tangible nature of the extensive information which it readily affords. The relations of hooping-cough and temperature having been given, I need not again refer to them, but in describing those of the other lines of disease, I shall, throughout, have in view the intention of further illustrating this relation.

First, in reference to the line of general mortality. It is quite clear that the general direction of this line is directly opposed to that of temperature—the highest mortality occurring at the season of lowest temperature, or winter, and the lowest mortality at the period of highest temperature, or summer. The highest mortality is observed about January, but varying from December to March, and in 1852, was so late as May, whilst the lowest mortality occurs almost invariably

in June and July. In the cholera year of 1849, and in that alone, the lowest mortality was observed so late as November—that is, after the epidemic had subsided—and may naturally be attributed to the lack of subjects of fatal disease. The most healthy period of the year is from April to November, except in such years as experience the recurrence of fatal epidemic diseases. It should also be remarked, that, for the most part, the most fatal seasons in a series of years, are such as have the lowest temperature, as was the case in the winters of 1847-8 and 1852-3; whilst, on the other hand, the periods which experienced the lowest mortality in a series of years, as 1848 and 1850, were marked with the highest degree of temperature. The months of highest temperature and lowest mortality are not usually the same, but, as in 1849, the latter is a month later than the former. Thus it was particularly the case when the temperature had somewhat suddenly increased; for when the temperature throughout the winter had remained somewhat high and stationary for some months, as in 1850-1, or when it had increased considerably in March and April, as in 1847, the monthly lowest mortality was in advance of that of highest temperature. In the latter case, it would seem that the long-continued high temperature became, beyond a certain point, a cause of mortality. On contrasting this statement respecting the general mortality with that of hooping-cough, several disparities will be observed sufficient to show that the cause of mortality from the latter disease is not identical with that of the general mortality.

In abstracting the returns for the three great classes of disease, I have comprehended the whole of each class, on the ground that, however diverse one number may be from another, they have essential points of resemblance; and also, because I could not venture to affirm that any one which I might wish to exclude had less connexion than any other with hooping-cough. The object is attained if they show, in general terms, the relation which they bear to hooping-cough.

The zymotic class, is remarkable from the opposition in

its lines with those of hooping-cough, since there are undoubtedly essential bonds of union between them. This opposition is the more remarkable in that hooping-cough is included in the line of zymotic disease.

In the zymotic class the lowest mortality is observed to correspond with the low temperature, and therefore with the beginning and the close of the year; whilst its highest mortality is observed in August or September, and therefore corresponds with the period of considerable, but not of the highest temperature. In no instance does its acme precede that of temperature, but it either corresponds with it, as in the cholera epidemic of 1849, or, as is more customary, immediately succeeds it. Its progress appears to be in cycles, having its origin or lowest point immediately after a severe outbreak of the disease, and thence remaining stationary for a period, but ultimately increasing in mortality by slow increments, until it again attains its maximum. There has been, as yet, no such yearly zymotic mortality since 1849, as was observed in the years immediately preceding 1849, but there has been a gradual increment since 1850. In all these various points, this great class differs from hooping-cough, and in its essential character is directly opposed to it. Indeed, there is not an instance during the seven years in which the lines of alternate increase and decrease of hooping-cough are not directly opposed to those of the great zymotic class; whilst, on the contrary, in almost all cases the zymotic lines and the lines of temperature tend to the same direction. This is a strong argument against the essential affinity between mortal cases of hooping-cough and the class under consideration. It is therefore certain, that our deductions in respect of hooping-cough are not weakened by any possible similarity between it and the zymotic class of diseases.

Directly opposed to the zymotic class is that of pectoral affections, for the lines of this class are in opposed waves to that of temperature, and in marked correspondence with those of hooping-cough. The highest point of mortality is almost invariably met with in January, and

corresponds accurately with that of the lowest temperature. In this latter respect the pectoral class differs from others, hooping-cough included, for its mortality keeps nearly even pace with the temperature. This is very strikingly manifested upon the Diagram. Its lowest mortality, too, is observed at the very months which have the highest temperature of the season, and thence remains nearly stationary during two or more months, or has a gradual tendency to increase. The months intervening between April and November, or December, are the least infected with this class of diseases, and in this respect, this class corresponds with the general mortality. The only noticeable distinction to be made between the lines of mortality from hooping-cough and chest diseases is, that whilst both invariably take the like direction, the former follows the latter in descending, and precedes the latter in ascending. The great similarity between hooping-cough and chest diseases, contrasted with the dissimilarity between the former and zymotic affections, cannot fail to induce us to regard them as most closely allied, and may almost suffice to induce us to enquire if they are not, in their morality, the same disease. It must not be forgotten, that hooping-cough, as such, is seldom fatal, and that the mortality really arises from its complications; if, therefore, we admit the evenness of the mortality of the two diseases, it would only be affirming, that in a great majority of the deaths from hooping-cough, the chest complication is the cause of the death, and it would leave the eventual nature of hooping-cough untouched.

The third great class of diseases, or the nervous, offers but unsatisfactory evidences of its affinity to hooping-cough, and that, perhaps, from the fact just alluded to, viz., that whatever hooping-cough may be, it is not usually mortal. The Diagram shows a remarkable uniformity and narrowness of limit in the range of this class of disease through each year, and through a series of years. The line scarcely, if ever, has a greater range than 50 cases, and throughout the whole year does not extend through one half that amount. It can, therefore, scarcely be influenced by the

change of seasons, and, consequently, can offer but little affinity to hooping-cough, the general mortality, zymotic, or chest affections. The highest point, little varied as that may be, appears to be during the cold season, and its lowest during the middle months of the year. Thus, on a review of the analyses of the mortality lines on the Diagram, we may affirm that the lines of hooping-cough do not precisely correspond with those of the general mortality; that they are directly opposed to those of the zymotic class; that they are greatly in accord with those of chest diseases; and lastly, that they have but little evident relation with those of nervous diseases. Thus we infer, that hooping-cough is a disease apart from those affections, and that any deductions made from its returns, cannot be weakened by any supposed resemblance between it and these classes of diseases. Further, we may affirm that mortal cases of hooping-cough disprove any alliance between it and zymotic disease, leaves it in doubt in reference to nervous diseases, and offers much support to an alliance with chest affections.

In order to exemplify the foregoing statements more clearly, I have compiled Table and Diagram III. These show the weekly average of the temperature, hooping-cough, and bronchitis, in the seven years already referred to, condensed into one year. By this mode, the ordinary variation is nowhere uninfluenced by temporary causes, and a more correct notion is given of the seasonal temperature and its influence on the mortality of the diseases in question.

The highest temperature occurred in the 28th week, and thence the temperature gradually and progressively declined to the end of the year, when it was at the lowest point, and its degree was precisely that observed in the first week of the year, Thus, the first and the last weeks of the year have the lowest, and 28th week the highest temperature. The progression and retrogression exhibited much uniformity, and from the 6th to the 11th week, the variations were more perceptible than at any other period of the year.

The mortality from hooping-cough attained its minimum (25 per week) in the 33d week, and continued low until the

47th week, when it suddenly increased to 37 and 42 per week, and terminated the year with 40 per week, the precise number with which it began the year. From this point it gradually increased until the 12th week, when it attained its maximum, and thence gradually, but with many variations, declined to its lowest point. In reference to its relations with the line of temperature, the following points may be noted. The two lines intersected each other in the 16th and 51st weeks, and from the 19th to the 49th week were directly opposed to each other. From the 49th to the end of the year, and from the beginning of the year to the 17th week, the lines, generally speaking, assumed a parallel and closely approximated, yet variable, course. There was an interval of five weeks between the highest temperature and the lowest mortality, but it must be mentioned, that the 2d week after the highest temperature, the mortality was nearly at its lowest point. Thus the mortality continued to decline for some time after the temperature began to slightly decline. It also continued low, not varying 5 cases per week, so long as the temperature continued above 48°, and thence it assumed a rapidly upward tendency.

The general parallel course between the lines observed at the beginning of the year is no evidence that the mortality was uninfluenced by the temperature, for at various parts the relation was very manifest. Thus, the downward tendency of the temperature in the 3d week, induced at the same time an upward tendency to the mortality, and so also in reference to the lower temperature of the 6th, 8th, and 10th weeks, for these were followed by increased mortality in the 7th, 8th, 10th, 11th, and 12th weeks. The increase of mortality, therefore, followed the diminution of temperature at an interval of one or two weeks, but the new tendency thus given did not subside on the instant, with an increase of the temperature, but continued for a period longer. Whenever, therefore, in the variations, the two lines run a parallel course, or may be seen between the 6th and 13th weeks, the true cause of the course of mortality is antecedent to that of temperature. The mortality never

moves in advance of that of temperature, but often retains the impetus for a time after the projectile force has been withdrawn. Thus the general parallelism above referred to, directly confirms the truth of previous statements when analysed into its weekly variations. If further illustration were needed, it would be afforded by the lines passing through the 30th to the 39th week, in which the downward tendency of mortality is continued for a time after the temperature had ceased to increase, and in which alternate increase and decrease resulted from variation of temperature in the two weeks antecedent. Indeed, so universal is this rule in its application, that throughout the whole year the variations may be safely explained by it. The line of temperature below which hooping-cough runs its most mortal course, passes through the 48°, both in its advance and in its retrocession.

I have selected bronchitis as a point of comparison, on the ground established by diagram II, viz., that a close affinity exists between the class of pectoral affections (excluding phthisis) and mortal cases of hooping-cough; and further, because of all pectoral affections, I was of opinion that bronchitis was by far the most common disease. The line of mortality from bronchitis is worthy of attention, both absolutely and relatively to hooping-cough. Its highest point is in the 49th week (142 cases per week), whence it rapidly descends to the end of the year to a point lower (16 cases per week) than at the commencement of the year, and continues high until the 11th week, when it suddenly and progressively declines to the 31st week, and is at its minimum (27 cases per week); and after remaining nearly stationary during 6 weeks, begins rapidly to ascend to the 37th week, and in 12 weeks reaches its maximum. Thus it is essentially a winter disease, and leaves so large a portion as the half of the year, at which its mortality does not attract attention. The chief points of contrast between this and hooping-cough are, that its variations attend more instantaneously upon temperature, and are to a much greater extent; that its highest mortality occurs at other times, and

that the mortality remains stationary at and ascends under a higher temperature, whilst its point of agreement is the general direction of its lines with hooping-cough, and opposition to those of temperature.

The following conclusions are a few of those which may be drawn from the foregoing communication:

1. In reference to its frequency:

In the London district the diseases which are more fatal are phthisis, pneumonia, bronchitis, typhus, convulsions, and scarlatina, in their order. In all England, in 1847, diarrhœa was added to this list, and their order varied. There was greater diversity in the great registration divisions, both as to the precedent diseases, and their order of mortality.

The proportion to the total mortality in London is 1 : 29·6. In all England in 1847, 1 : 45·7, and varying in the great divisions from 1 : 28·1 in the eastern, to 1 : 94·8 in the south-western. It is as 1 : 1824 of the total population.

The most fatal years from 1844 to 1853, in London, were 1849 (45 per week), and 1853 (50 per week); and although both these years had high general mortality, the increased mortality from hooping-cough was not due to that circumstance.

The lowest mortality was observed in 1844 (25 per week), and that did not correspond with the general mortality.

The deductions in reference to hooping cough do not correspond to the general mortality. They are directly opposed to the zymotic class, and have little relation to the nervous class, but exhibit a remarkable correspondence with the pectoral class (excluding phthisis). This latter fact indicates a close analogy between fatal cases of hooping-cough and chest affections.

2. In reference to age:

It is a disease essentially of the period of dentition of the first series, and under æt. 1 year is the most fatal of all diseases. It thus differs from all other members of the zymotic class.

3. In reference to sex:

The mortality is more prevalent in females at every period of life, and this prevalence increases as life advances; but it does not thence follow, that the disease itself is more prevalent in that sex. But if it be so, it is probably due to the susceptibility to impression, and the power of passive endurance which characterise the organization of females.

4. In reference to temperature:

The degree of temperature, and the number of deaths, are in the inverse ratio to each other. The greatest mortality is observed in the 1st quarter, and also in the winter half year, and the least mortality in the 3d. quarter and the summer half year. The maximum month is about April, and the minimum about August, and the mortality passes from the latter to the former and back again in uniform waves. The highest temperature precedes the lowest mortality by about a month, and an excess of it does not produce excessive mortality from hooping-cough. The line of temperature separating high from low mortality is 48°. The waves of the greatest intensity of mortality occur every second year, being then 76 and 74 cases per week, in contrast to 47 and 52 cases per week observed at the highest mortality in the alternate years, or those of recession.

After a severe outbreak of the disease, there is diminished intensity, and lessened temperature does not then produce its ordinary ill effects. As the year of recession leads into that of intensity, the intensity becomes so great as to move *in advance of*, and not in the rear, of the lines of temperature, contrary to the established rule.

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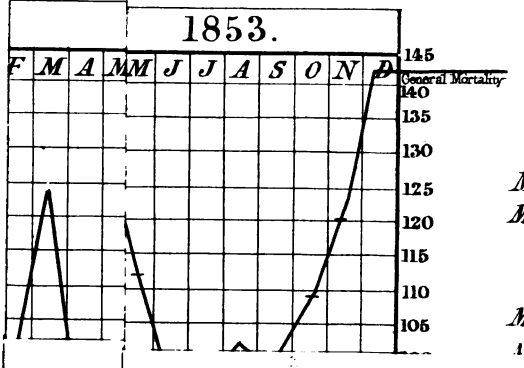

Temperature.

Max. . . . *July.*

Min. . . . *January.*

Hooping Cough.

Max. . . *March.*

Min. . . . *August.*

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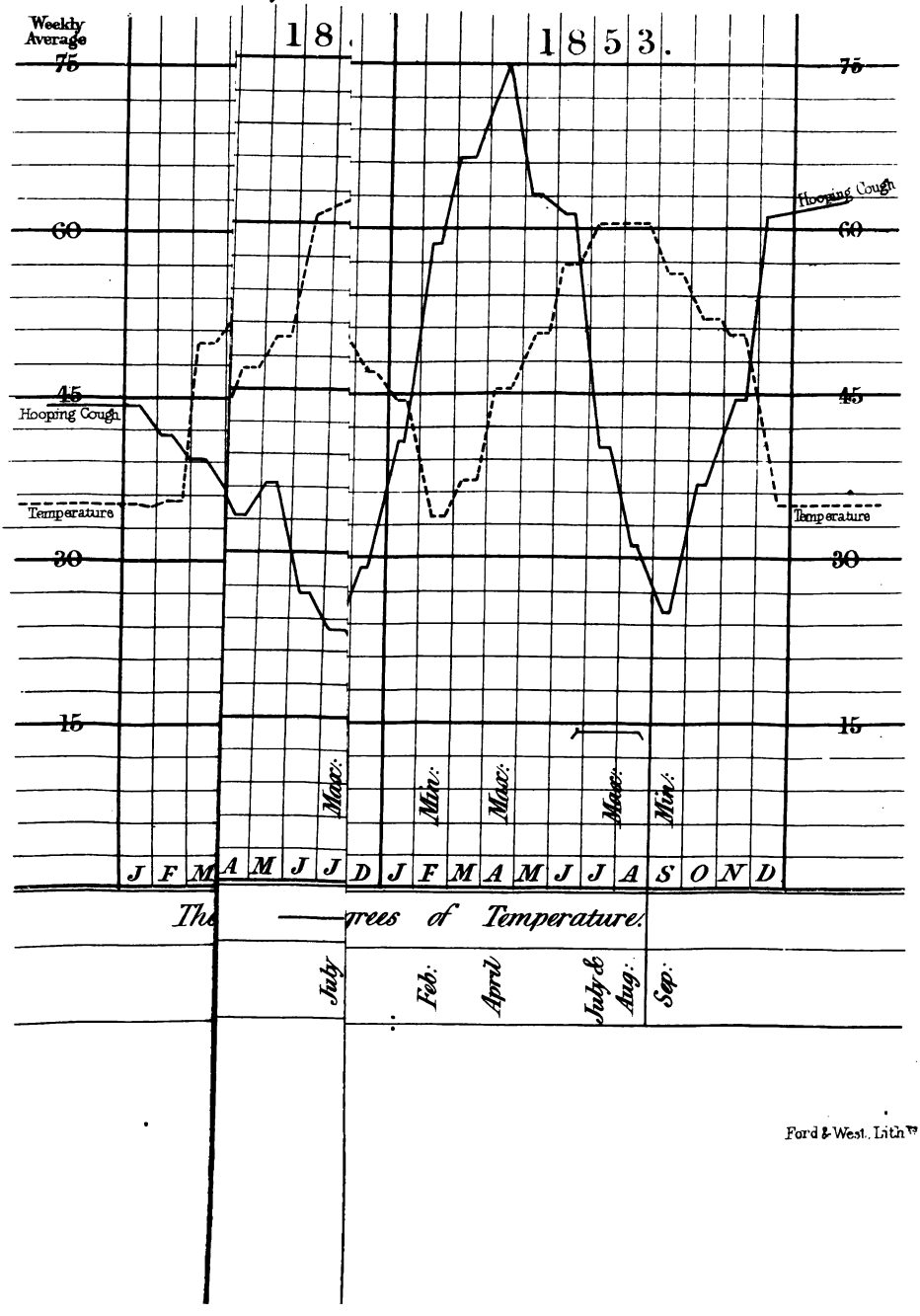

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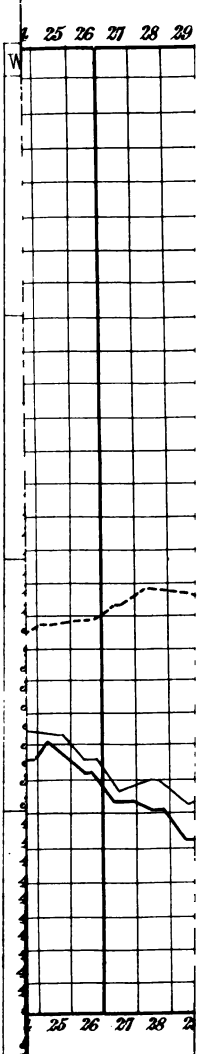

Temperature.

Maximum ... *28th week.*
Minimum ... *1st & 52nd do*

Hooping Cough.

Maximum ... *12th week.*
Minimum ... *33rd do*

Bronchitis.

Maximum ... *2nd week.*
Minimum ... *31st week.*

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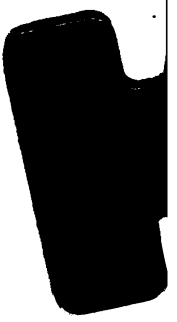
END OF VOL. XXXVII.

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